THE
LARYNGOSCOPE.

VOL. XLIII DECEMBER, 1933. No. 12

NASAL DEFORMITIES AND THEIR REPAIR.

DR. V. H. KAZANJIAN, Boston.

Nasal deformities, in general, may be divided into two large groups. The first comprises all the major deformities caused by destruction of the whole or part of the nose. The second includes minor deformities and malformations of the nose. While, in the second group, the loss of the substructure of the nose may contribute to the defect, the majority of such deformities are due either to distortion of the different parts of the nose or to a super-abundance of normal tissue.

The aim of rhinoplasty is to establish perfect functional as well as desirable cosmetic results. Owing to surgical limitations, however, and also to the various handicaps peculiar to the individual case, our measure of success is relative only. In gross deformities, the major surgical problem centers round the ability to obtain enough tissue from the immediate neighborhood of the defect or, if it must be so taken, from distant parts. For an extensive deformity of this type we do not hope to reproduce a perfectly natural looking nose; we are satisfied if the patient ceases to be conspicuous because of his appearance. In minor defects and malformations one may justly be more critical as to the degree of success. For, in this instance, the question of supplying new tissue does not usually exist; if it is necessary, however, it is not an acute problem. On the

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication April 15, 1933.

other hand, operative technique is more delicate, and for that reason the operation, whether it is to remove, to add, or to readjust tissues of the nose, must be done very cautiously with a minimum of outside scars.

In this short paper I propose to discuss some of the problems connected with the repair of major deformities that are associated with a definite loss of nasal tissue arising from accidents, disease or operations for malignant tumors. The loss of tissue may be varied in size and location; it may involve the skin only, or it may include the bony and cartilaginous framework as well as the inner surface of the nose. Obviously, there are many degrees of nasal deformities, but for descriptive purposes I shall divide them into the following groups:

1. Loss of the skin only;
2. Loss of part of the framework, skin and mucous membrane;
3. Loss of framework only.

The first group where the skin only is lost is attributable to operations for malignant disease or injuries, especially automobile accidents. The severity of the case is proportional to the size of the missing tissue, and whether or not, the tip and the nostrils also are involved, causing distortion or stenosis of the nasal passages.

In comparatively minor defects one is able to repair the nose by utilizing the remaining skin to fill the gap, provided that the patient has a nose large enough to permit a diminution in size. (See Case 4, Figs. 11 and 12.)

In general, these defects have to be prepared by utilizing skin grafts taken from surrounding areas.

Skin grafting over the nose in the form of a full thickness graft has certain advantages, but it also has some serious disadvantages. It is a simple procedure, of course, and under proper conditions is generally successful. Furthermore, the length of time involved in hospitalization and the convalescing period are made shorter. Unfortunately, however, we are not always sure of a perfect result because the skin graft occasionally absorbs and contracts too much and the color of the transplant sometimes does not blend well with the surrounding skin. In either case the cosmetic result is usually poor, and for these reasons I seldom resort to skin grafting to cover a defect of the nose.

Our next method of repair is to utilize a pedicled graft. By the use of delayed flaps we can transfer skin from the arm, chest, neck, back of the ear, scapula region or from the forehead. From the point of view of simplicity as well as appearance, the forehead flap has much in its favor, the only objection being that it results in an additional scar over the forehead. To my mind, however, this objection is slight in comparison with the advantages to be gained by its use.

Tube flaps from the arm, chest, etc., may be used advantageously if the plan involves the repair of other parts of the face in addition to the nose, such as deformities resulting from extensive burns.

Cases 1 and 2: The second type of deformities involves the framework and mucous membrane. Such deformities may result from disease conditions of a destructive character, such as lupus or lues, or from the radical operation for a malignancy.

Where the case does not result from a malignancy, partial or total rhinoplasty is within operative possibilities, and one does not hesitate to advise a patient in good physical condition to undergo several operations. Where a malignancy enters the picture, this procedure is not always a good operative risk because of the uncertainty of a cure, advanced age, the danger from too early closing of the wound, and the frequent impossibility of indefinite hospitalization. For the repair of such cases, however, if conditions are favorable, I again give preference to the use of a forehead flap. Our problem here is more complicated because, in addition to the skin, we are obliged to supply the lining membrane and also restore the contour of the nostril if the lobe is destroyed.

For the lining membrane, we have the choice of utilizing a flap of skin from the side of the cheek or covering the inner surface of the forehead flap with a skin graft. Our choice for each individual case is dependent upon the type of defect.

Cases 3, 4 and 5: Of course, in all this type of work one must not overlook the definite uses of prosthetic noses. When one is obliged to excise the greater part of the nose for the removal of cancerous tissue, an artificial nose is often the most satisfactory solution. These noses are generally made of vulcanite rubber. They are painted with celluloid paint to harmonize with the color of the skin and are retained in position by various devices. While they are artificial and carry with them a certain amount of repul-

sion, they are actually useful even as a temporary expedient, since when well modelled and painted, they are quite inconspicuous. Their greatest advantage lies in the fact that they give the surgeon an opportunity for more thorough inspection of the wound from time to time. (See Case 6.)

To restore the missing sections of the nostrils and alae of the nose is one of the problems of rhinoplasty. The contour of the nasal opening is controlled by the lower lateral carti'age covered with skin and mucous membrane, and when part of it is destroyed it is difficult to restore it to its former contour with transplanted tissue. A fair degree of success is acquired by using the following methods:

1. When a forehead flap is made, the distal borders are folded in sufficiently to supply epithelial tissue to the borders of the nostrils.

2. A flap from the side of the face may be turned in to form the inner surface while a forehead flap covers the outer surface.

3. The inner surface of the forehead flap may be covered with skin graft previous to transposing over the nose.

None of these are ideal because the borders of the nostril are made unusually thick. Yet in the third method we have less difficulty, a factor which perhaps renders it more desirable.

In minor defects of the nostril, one is able to utilize the defective edge by bringing it forward and repairing the resulting distal gap with a transposed flap. Then also in selected cases I have been able to utilize part of the septum to repair the nostril, as exemplified by the following case:

Case 7: The patient, a young woman, had lost the greater part of the ala nasus in childhood (see Fig. 21). The repair was made in the following manner: A rectangular incision was made through the septum, beginning from behind the columella down to the base of the septum, a distance of about 15 millimeters, where a distal vertical incision to the upper border of the septum was made. This septal flap, which received its blood supply from above, was sutured to the defective borders of the nostril, and was held in position by a small gutta percha shield which was made to fit the nostril accurately. Three weeks later the upper end of the septal flap was severed from its base and sutured near the tip of the nose. My original plan was to remove the mucous membrane covering from the external surface and replace it with a skin graft, but as the

general appearance was not conspicuous, it was left alone. (See Fig. 22.)

Case 8: Deformities involving the loss or distortion of the framework of the nose will naturally cause flatness of the bridge, the severity of which depends upon the amount of tissue lost. This deformity may originate from a variety of causes, but what interests us most in this paper are traumatic injuries, and other major

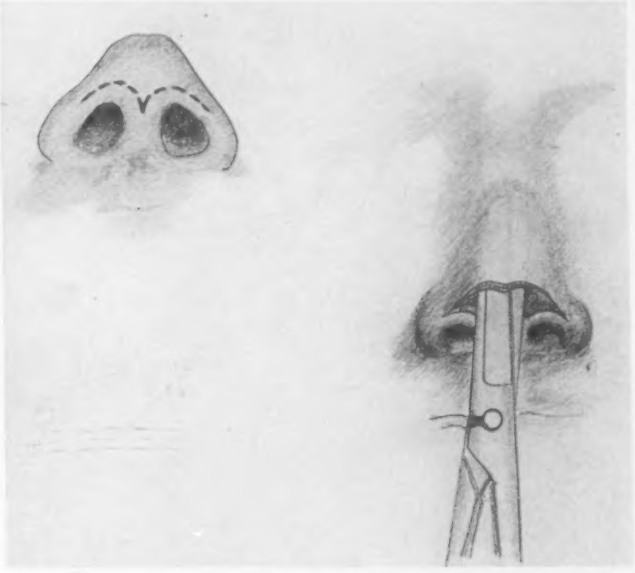


Fig. 30.

deformities following osteomyelitis of the nasal bones or syphilis. In such deformities we assume that the skin of the nose is intact and the depressions are merely due to the lack of proper supporting tissue. For the treatment of such conditions one is obliged to resort to transplantation of bone or cartilage.

Living transplants most suitable for the repair of the bridge of the nose are cartilage or bone. The relative merits of bone and cartilage are discussed quite extensively in the literature and a considerable amount of experimental work has been made along this line. As a

result cartilage is favored by a great many. I have no intention to engage in a discussion or argument on this question. In my own experience I have used both bone and cartilage for this type of transplantation, and have achieved a reasonable success with each. The whole question devolves around the particular needs of each patient in relation to the nature of his deformity and attendant circumstances.

Not infrequently deformities resulting from accidents may have indurated scars over the nose, and at times definite loss of skin tis-

Fig. 31.

sue, or even perforation into the nasal cavity. In such cases one is obliged to excise the scars, repair the defect of the skin and prepare the area for the introduction of the transplant later on.

Case 9: In operations of the nose for either transplantation of bone or cartilage, it has been my custom for many years to make a "bat wing" incision over the tip of the nose, and through this opening, the skin is tunnelled from the framework of the nose. (See Fig. 30.) My reason for an external incision, especially over the tip, is that it allows for a greater exposure of the field, thus simplifying the operation and giving the operator more latitude for the correction

of secondary defects, and it effectively isolates the field from the nasal cavity. Contrary to expectations, it leaves an imperceptible scar.

Deformities caused by acquired syphilis are fortunately rare, but when they occur they usually present a complex picture. The loss of framework combined with adhesions of the mucosa of the nose

Fig. 32.

may cause complete collapse of its contour. Also there is often a definite destruction of tissue, especially about the nasal passages.

Examination and Diagnosis:. In all rhinoplastic operations it is important to make a careful examination of the component parts of the nose in order to determine the nature and extent of the deformity. To do this one must keep constantly in mind the detailed

anatomy of the normal nose and its general contour in relation to the face. With a definite image of the normal we are in a position to see clearly the various deformities caused by distortion or destruction of the various parts. In traumatic cases one often finds that there is no actual loss of tissue, but hastily performed primary suturing gives an example of one kind of deformity.

One must not be content with a visual examination alone. Photographs and plaster casts of the face must be taken and these will provide not only a valuable working model, but will also serve as a permanent record.

Postoperative Treatment: One of the most important factors of the postoperative care following rhinoplasty is the use of a suitable

Case 1, Fig. 1. Case 1, Fig. 2.

splint for protection against possible trauma, for the support of the newly transplanted tissue and for preventing the collection of blood and serum under the subcutaneous tissues.

For this purpose a piece of flat tin, gauge 23, shaped like an hour glass, is bent so that the lower part conforms to the general shape of the nose and the upper part rests flat against the forehead. This piece of metal serves as a tray for a small quantity of dental modelling compound. When the contrivance is adjusted in proper position, the compound comes in contact with the nose and, on account of its plasticity guarantees an equalized pressure on all sides. The whole appliance is retained by two strips of surgeon's plaster, one passing across the forehead at the top and the other passing over the cheeks beneath the eyes at the bottom (see Figs. 31 and 32). It

is not necessary to exert more than moderate pressure, and the splint or dressing should not be disturbed for at least two days. When it is removed it will be found that the nose is surprisingly free from inflammation and edema. This method I have used with considerable success for a number of years.

Case History 1: Mr. W. This patient reported on June 24, 1931, relative to a lesion of the nose which had started as a pimple three years previous. He had received X-ray treatment with no improvement.

Examination showed a sharply outlined ulcerated area 2 x 1 cm. in diameter in the middle of the nose close to the bridge. The base

Case 1, Fig. 3.

of the ulcerated area was whitish and moist, while the borders were sharp and indurated. The report of the specimen taken for biopsy was "basal cell carcinoma." Excision was advised (see Fig. 1).

On Sept. 12, the patient was operated upon and the cancerous lesion excised with a good margin of healthy tissue (see Fig. 2). He made a normal recovery and the plastic repair was begun on Oct. 1. In the first operation a forehead flap was prepared; this flap was later transferred to cover the deformity of the nose. The repair was completed in four operations performed under local anesthesia and extended from Oct. 1 to Jan. 4, 1932.

I saw the patient on Jan. 6, 1933. He was in good physical condition, the plastic result was good and there was no evidence of recurrence (see Fig. 3).

Case History 2: P. K. This patient was first seen by me on May 27, 1929. He gave a history of having acquired syphilis ten years previously. The lesion spread over the face four years ago and had been treated with radium ever since. Examination showed dense sclerosed tissue over the entire surface of the nose as well as part of the right side of the face. There were islands of ulcerated sores scattered through the scar tissue. The lesion on the whole seemed to be superficial, and there was no invasion into the deeper layers of the nose. Biopsy report was "hair matrix carcinoma" (see Fig. 4). On June 4, 1929, the patient was operated upon under local anesthesia and a forehead flap prepared. Following this, on June 15,

Case 2, Fig. 4.

Case 2, Fig. 5.

he was again operated upon under local anesthesia, and all the malignant tissue was dissected out. The forehead flap was brought down to cover the raw area (see Fig. 5). On July 10, the unused part of the forehead flap was transferred back to its normal position and the raw area on the forehead was covered with a full thickness skin graft. The patient made an uneventful recovery until three years later, when he had a recurrence of cancer about both inner canthi. He was again operated upon on Jan. 4, 1932. During the operation it was found that the cancerous tissue had extended over the root of the bridge. Free excision was made and the defect was closed by two triangular flaps from above the eyebrow (see Fig. 6). As the patient was leaving the city to go west, he was advised to seek medical attention as soon as he became located. Eventually he consulted Dr. Joseph Beck.

Case History 3: W. G. This patient was operated upon by Dr. Charles Lund in March, 1929, for adenocarcinoma of the right maxillary sinus, palate and lip.

Case 2, Fig. 6.

Case 3, Fig. 7.

Case 3, Fig. 8.

Examination (see Fig. 7) showed that the patient had lost part of the right side of the nose, upper lip and the anterior third of the palate. The mouth was edentulous, and there was an old un-united fracture on the right side of the lower jaw. The wound was free from any suspicion of malignant disease. At this time a vulcanite plate was made to fit over the upper jaw.

Prior to plastic operations for the repair of the lip, a temporary upper denture was made which was retained in position by a finger-like projection of the vulcanite towards the floor of the nose.

Seven operations were performed under local anesthesia, extending from April 28, 1929, to Jan. 25, 1930.

The lip was repaired first by utilizing the remaining part of the upper lip on the left side, and second by taking a rectangular flap from the right side of the lower lip, using the full thickness of the cheek.

Case 3, Fig. 9.

Case 3, Fig. 10.

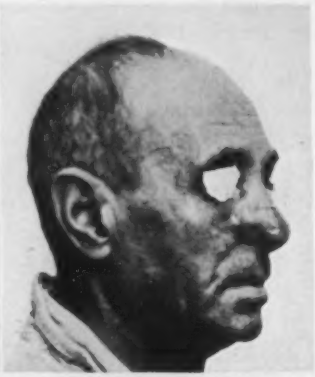
For the repair of the nose a skin graft was introduced to the under surface of a forehead flap; this flap was later brought down to close the nasal defect (see Figs. 8-9).

After leaving the hospital a new dental plate was constructed to fit the patient's upper jaw, thus making it possible for him to talk distinctly (see Fig. 10).

Case History 4: Mr. C. This patient had a cancerous lesion on the right side of the nose about 1 cm. in diameter (see Fig. 11). He was admitted to the hospital and the lesion was excised under local anesthesia in 1925. The nose was repaired by (1) using a suitable flap of skin from the side to form the inner layer, and (2) reducing the size of the entire nose and utilizing part of the skin from the left side of the nose to cover the outer surface of the defect (see Fig. 12).

He was not seen again until 1930, although follow-up cards had been sent from the hospital asking him to report for examination at various intervals. At this time examination showed that there

Case 4. Fig. 11.



Case 4. Fig. 12.

Case 4. Fig. 13.

was an extensive recurrence of the carcinoma involving the entire right side of the nose, nasoantral wall, maxillary antra and side of the face. He was operated upon again; the operation consisted of the excision of the side of the nose, and part of the tissues of the face (see Fig. 13). The maxillary sinus was cleaned, and the entire

nasoantral wall was removed. The right upper and lower eyelids were sutured together to prevent the possible distortion of the lower lid from resulting scars. The deformity was covered by a prosthesis which was made at the Harvard Dental School, and consisted of one-half the nose and an extension covering the opening of the face (see Fig. 14).

Case History 5: Mr. C. This patient reported at the skin clinic of the Massachusetts General Hospital on Dec. 31, 1925, relative to a lesion of the nose of six years duration. He was referred to the tumor clinic with a diagnosis of Lupus Vulgaris, Epithelioma. He was admitted to the hospital.

Case 4, Fig. 14.

Examination showed the end of the nose reddened and scarred. The septum at the ostia of the nostrils was eroded, and the erosion extended to the mucous membrane on both side; the septum was partially eroded and the nares filled with scabs. The tonsils were enlarged, teeth in poor condition with marked pyorrhea. He also had an otitis media on the right side which necessitated a paracentesis on Jan. 6. Microscopic examination of the nose showed carcinoma while the report of the specimens taken from the lip and vomer was negative.

On Jan. 27, 1926, the patient was operated upon and the cancerous lesion of the nose excised. On April 3, he reported at the

hospital for plastic repair, but he was advised to wait at least a year. In the meantime he was referred to the Harvard Dental School for the construction of a temporary nasal prosthesis.

Case 5, Fig. 15.

Case 5, Fig. 16.

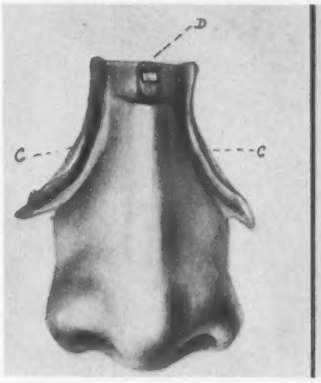
Case 5, Fig. 17.

On March 29, 1928, he was again admitted to the hospital. His general physical condition was improved and there was no evidence of recurrence of the disease. The first operation for plastic repair of the nose was performed on April 3, and was followed by 5

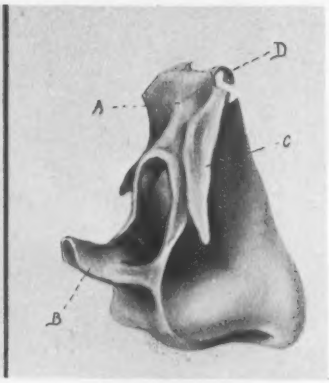
others on May 8, June 28, July 13, and on May 7 and 15, 1929. He was discharged on May 22, 1929, and has not reported since (see Figs. 15, 16 and 17).

Case 6, Fig. 18a.

Case 6, Fig. 18b.



Case 6, Fig. 19, c, d.



Case 6, Fig. 19, a, b.

Case History 6: Mrs. S. This patient was seen on Feb. 20, 1930. Examination showed extensive squamous-cell carcinoma involving the greater part of the nose. The lesion had already invaded the septum and mucous membrane of the nose (see Fig. 18A). On

April 3, the patient was operated upon under general anesthesia. With an electro-surgical knife the entire diseased tissue was excised with a good margin of healthy tissue, involving, in addition to the

Case 6, Fig. 20.

Case 7, Fig. 21.

bony and cartilaginous structures, the anterior part of the septum and upper half of the upper lip. The entire surface of the wound was cauterized with diathermy. On Sept. 9, the wound of the face was completely healed; the upper lip, however, consisted of a thin strip

of tissue not over 1 cm. in width and was bound down by adherent scars. An operation in order to remedy this condition was advised.

Under local anesthesia the upper lip was elongated to the proper size by making an oblique incision throughout the entire thickness of the lip and overlapping the flaps until the proper size was acquired (see Fig. 18B). On Oct. 2, an artificial nose was constructed from vulcanite rubber. The main features of the prosthesis were as follows: (1) the inner surface (see Fig. 19A base) fitted over the face accurately and covered as much space as was consistent to the shape of the nose; (2) the prolongations of the restoration (see Fig. 19B) rested upon the floor of the nose, the purpose being to

Case 7, Fig. 22.

prevent the artificial nose from sliding down upon the lip; (3) the lateral grooves as well as the metal clasp over the bridge (see Fig. 19C-D) fitted accurately to the bridge and the rims of the spectacles so that when the nose was worn there was an even pressure from the spectacles to the upper half of it against the tissues of the face. The nose was painted to match the color of the skin with celluloid paint.

The patient was seen as recently as Nov., 1931. She was wearing the restoration comfortably (see Fig. 20).

Case History 8: Mrs. S. This patient was injured in an automobile accident on July 2, 1930, in which she suffered laceration of the tip of the nose and upper lip.

An examination of the patient on Sept. 11, 1930, showed (1) a distortion of the upper lip caused by a thick band of scar tissue which extended from the base of the nose to the vermilion border

Case 8, Fig. 23.

Case 8, Fig. 24.

Case 8, Fig. 25.

of the lip; this band caused contraction of the lip, showing the upper teeth; (2) the tip and the lower part of the nose, especially on the left side, were destroyed (see Fig. 23). It was decided to repair the upper lip first.

On Jan. 24, 1931, the patient was operated upon under local anesthesia. The lip was repaired and at the same time a forehead flap was prepared with a skin graft lining over an area large enough

Case 9, Fig. 26.

Case 9, Fig. 27.

Case 9, Fig. 28.

Case 9, Fig. 29.

for future use in repairing the tip of the nose (see Fig. 24). In successive stages this flap was brought down and utilized to repair the tip, the columella and left side of the wing of the nose. The exposed area of the forehead was covered with a full thickness graft (see Fig. 25).

Case History 9: Mr. L. This patient was injured in October, 1931, in a motorcycle accident, in which he received severe injuries

to the skull, nose and bones of the upper part of the face. An examination, on May 5, 1932, showed the nasal bones badly depressed and irregular, the lower part of the nose distorted and a perforation leading into the nasal cavity about 2 cm. below the frontal eminence. There was also a scar extending from the base of the ala of the nose on the right side to the corner of the mouth (see Fig. 26). The repair in this case consisted of two operations. In the first a submucous resection of the septum was done and the perforation closed by bringing a diamond-shaped flap from the forehead (see Figs. 27 and 28); the second operation consisted of a tibial graft inserted to the bridge of the nose. The patient made an uneventful recovery (see Fig. 29).

475 Commonwealth Ave.

BOVINE CARTILAGE IN CORRECTION OF NASAL DEFORMITIES.*

DR. PHILIP S. STOUT, Philadelphia.

Many and varied are the substances suggested to use for the correction of nasal deformities. I have used ivory, autogenous bone and cartilage from the rib resection or cartilage from the nasal septum,

Fig. 1. Beef scapula showing cartilage (above) separated from its attachment. Some of the flesh remains on the cartilage.

and various other substances, with the usual advantages and also the usual disadvantages, which may outweigh the advantages of these methods.

*Read before the West Philadelphia Medical Society, Jan. 21, 1933.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, July 1, 1933.

In 1924, at a meeting of the American College of Surgeons in New York, I met a surgeon from the Southwest, whose name I do not recall, who told me that he was using bovine cartilage for nasal deformity cases with uniform success. This impressed me, but I must admit that I was afraid to try it in Philadelphia, thinking that in the southwest the people might tolerate something which the people in the East might not. In short, I was afraid of an allergic manifestation caused by this beef or bovine cartilage.

Some three or four years ago I found that Dr. Edmund B. Spaeth, an eye surgeon at the University of Pennsylvania Graduate Hospital, Philadelphia, was doing eye socket reconstruction with beef

Fig. 2. Bovine cartilage over ruler (ruled in inches) showing size of cartilage. Beneath ruler, cut piece of cartilage, showing thickness and length of pieces you can easily obtain.

cartilage. This recalled the incident in New York some years earlier and I began to correct nasal deformities with bovine cartilage. Since then others also have used it in Philadelphia; among them have been Drs. Lewis, Lott, Fox, Kustin, Davis and others. Some like it, others do not.

The simplicity of the thing is what makes me feel we should give it a thorough trial before condemning it. So far it has proven about as satisfactory as any other method or material I have used. In the majority of cases it is very satisfactory, but if infection occurs, as

in any other method, the implant must be removed, but later the nose may be operated on and a fresh piece inserted. So far I have not found any allergic manifestations, although it is not impossible that such might occur. The cartilage is from part of the scapula of the average ox and in size, when flattened out, is some 7½ inches long by 3½ inches wide and of thickness varying from ½ to 1½ inches (see illustrations). You can see that this is excellent, because you can get a piece any size or thickness and it can be obtained from any butcher. I believe it is usually cut out of the meat and discarded. To prepare it, clean off all of the flesh from the cartilage, then cut into sections small enough to go into a wide-mouthed pint bottle, then cover it with a 10 per cent formalin solution. After three or four days it is put into a 4 per cent formalin solution, where it is allowed to remain until you desire to use it. It is then defor-

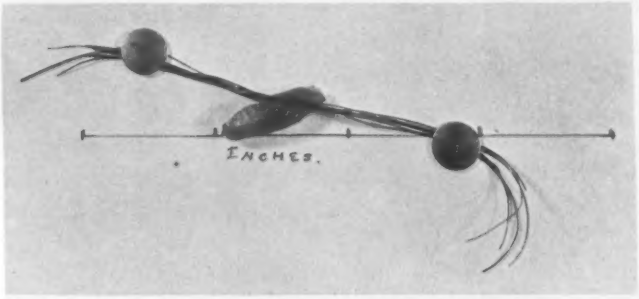


Fig. 3. Implant of bovine cartilage, removed after nine months, showing canal through cartilage. Six horse hairs were easily passed through the opening. There was no opening in the implant when it was inserted.

malized by putting it into sterile water and changing the water every day for three days. This deformalized cartilage is now ready to use. It can be used with or without the perichondrium. I have used it both ways and the results are about the same. If it is left too long in the formalin solution, it becomes slightly brittle.

After using this method the nose swells somewhat, which is also true when other substances are used.

I have had a chance to observe what may happen to the cartilage by the fact that an implant was knocked to one side of the nose nine months after implantation. I removed it (see illustration). A channel had been formed through the implant and blood was in this channel. After nine months the implant was slightly softer than

when inserted. It has been suggested to perforate these implants before inserting them.

As far as final results of nasal deformity correction are concerned, they are about the same as with any other method I have used. Moving picture demonstration.

269 South 19th Street.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Boston, Mass., Sept. 16, 1933, during the meeting of the American Academy of Ophthalmology and Otolaryngology. Seventy-nine candidates were examined, of which thirteen were conditioned or failed.

The Board will hold an examination in Cleveland, Ohio, June 11, 1934, during the meeting of the American Medical Association. Also early in the summer (date is not set) an examination will be held in Butte, Mon., in connection with the meeting of the Pacific Coast Oto-Ophthalmological Society. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb., for proper application blanks.

PARAPHARYNGEAL ABSCESS FOLLOWING TONSILLECTOMY. EXTREME HYPERPYREXIA. OPERATION. RECOVERY.

Dr. Joseph G. Gilbert, Brooklyn.

Pharyngeal abscess is occasionally seen as a sequel to tonsillectomy. The following report is of interest because of the hyperpyrexia which accompanied the condition. Such cases of parapharyngeal abscess have been observed by many laryngologists. Dean,[1] Glogau,[2] Claus,[3] Herrman,[4] Gunther,[5] Shapiro[6] and others have reported their experiences.

Case Report: A. N., white female, age 30 years, was operated upon by the writer on Aug. 30, 1931. The tonsils were removed by dissection and snare under novocaine infiltration anesthesia. Two days after the operation she noticed a swelling below the angle of her left jaw. This was associated with a slight rise in temperature. Examination six days after tonsillectomy revealed a healing right tonsillar fossa. The left tonsillar fossa contained a slight amount of purulent exudate and the posterior pillar on that side was thickened. The pyriform sinuses and the larynx were negative. An indurated swelling measuring 2 inches by 2½ inches was present in the region of the jaw and sternocleidomastoid muscle on the left side. The skin over this swelling was tender but not red. Her temperature was 102.2°. On admission it was considered that she had an infection of the parapharyngeal space (pharyngomaxillary fossa). Because fluctuation could not be felt and the patient seemed to be in good condition, conservative measures were employed. The treatment consisted of warm saline throat irrigations and the application of ice compresses to the neck. The temperature and the mass in the neck remained the same for the succeeding four days. On the tenth day after the tonsillectomy, the patient had a severe chill, which lasted about twenty minutes. The temperature rose to 108.8°. The temperature reading was verified with several different thermometers. At this time the patient was irrational.

Operation: With ethylchloride spray anesthesia an incision was made over the swelling along the anterior border of the sterno-

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 30, 1933.

cleidomastoid muscle. A clamp was inserted into the anterior part of the parapharyngeal space and directed toward the tonsillar fossa. A large abscess cavity containing fetid pus was encountered deeply placed in this space. A rubber dam drain was placed in the lower angle of the wound. Palpation of the tonsillar fossa, at the time of operation, revealed no communication with the abscess cavity.

The patient's condition improved rapidly following the incision and drainage. The temperature gradually reached normal on the fifth day after the operation. Her recovery was uneventful. A blood culture taken at the height of her chill was negative.

Follow-up for more than a year shows that she is in good health and has suffered no ill effects from her infection.

The abscess was undoubtedly caused by the injection of the local anesthetic. Organisms were apparently introduced into the parapharyngeal space through the superior constrictor muscle and buccopharyngeal fascia. The fetid pus which was encountered would indicate that we were dealing with an anaerobic infection.

At operation the type of incision and the question of ligating the internal jugular vein were considered.

The external swelling and tenderness and the negative intraoral and laryngeal findings suggested an incision parallel to the anterior border of the sternocleidomastoid muscle. An external incision with drainage of the prestyloid compartment of the parapharyngeal space was carried out. In some cases of parapharyngeal abscess, the suppuration points in the tonsillar fossa. These can be drained by carefully incising the superior constrictor muscle through the tonsillar fossa.[8, 9]

Because of the chill and hyperpyrexia, it was our belief that we were dealing with either a transient bacteremia or a mural thrombosis of the internal jugular vein. The condition of the patient and the fact that we had evacuated a localized abscess caused us to defer ligation of the jugular vein. The patient's condition improved rapidly after the simple incision and drainage, so that nothing further was indicated. Waldapfel[10, 11] has shown that in postanginal pyemia there are abscesses, about the tonsil, in the parapharyngeal space. He drained these abscesses, and paid little attention to the veins. His cases went on to recovery without ligation. He further notices that in those cases where he was unable to drain the abscess properly, the patients died in spite of ligation and resection of the veins.

It might be stressed that the course of a parapharyngeal abscess should be observed by direct examination as well as by external observation of the structures.

SUMMARY.

A case of parapharyngeal abscess which followed a tonsillectomy performed under local infiltration anesthesia is reported. The unusually high temperature (108.8°) is of special interest in this case. The importance of draining the localized abscess before it involves veins and other structures is stressed. In this case ligation of the jugular vein was not done. It is likely that many other cases of the same nature can be cured without ligation.

BIBLIOGRAPHY.

1. DEAN, L. W.: Severe Sepsis Following Tonsil Operations. THE LARYNGOSCOPE, 20:739, 1910.

2. GLOGAU, O.: Abscesses Descending from the Upper Air Passages Into the Neck and Mediastinum. Typical External Operation. Combined with Prophylactic Mediastinotomy. *Tr. Amer. Acad. of Ophthalmol. and Otol.*, p. 393, 1922.

3. CLAUS, H.: Achtundzwandig Fälle von Pyämie Nach Angina. *Ztschr. f. Hals, Nasen u. Ohren.*, 21:114, 1928.

4. HERRMAN, A.: Zur Pathogenese der tiefen Halsphlegmone Nach Tonsillecktomie. *Ztschr. f. Laryng. and Rhinol.*, 17:166, 1929.

5. GUNTHER, K.: Vier Jahre Tonsillektomie an der Wurzburger Universitäts Klinik. *Ztschr. f. Laryng. and Rhinol.*, 17:191 1929.

6. SHAPIRO, S. L.: Deep Cervical Infection Following Tonsillectomy. *Arch. Otol.*, 11:701, 1930.

7. See reference No. 6, p. 708.

8. See reference No. 2, p. 410.

9. See reference No. 6, p. 725.

10. WALDAPFEL, R.: Die post-anginäse Pyämie. *Ztschr. f. Hals, Nasen u. Ohren.*, 23:178, 1929.

11. WALDAPFEL, R.: Post-Tonsillitis Pyemia. *Tr. Amer. Acad. Ophthal.*, 33:291, 1928.

659 St. Marks Avenue.

ELECTRICAL DISTURBANCES IN THE COCHLEA PRODUCED BY SOUND.*

Dr. John Guttman, New York.

The structure of the ear is fairly well known. The function of the ear, however, the perception of sound, is still a source of numerous controversies.

Textbooks on otology generally hold that sound sets into vibration mechanically, by air pressure, the drum membrane, the ossicles, the lymph fluid, the basilar membrane, the hair cells, and by continued impact, the cochlear nerve. The nerve then conducts these vibratory stimuli to the cortex of the temporal lobe, where the conscious hearing perception takes place.

What role the different structures of the ear play in the physiological process of hearing is widely debated. It has not been determined what specific function to attribute to the intraauricular muscles. Great divergence of opinion exists regarding the mode of transmission of sound from the basilar membrane to the auditory nerve and to the cortex of the brain. We find attempts at explanation in the Helmholtz resonance-place theory, in the Ewald sound pattern theory, and in various modifications of these theories. The "all or none" nerve theory widened the controversies regarding the hearing perception.

In the last few years attempts were made to throw some light on the physiology of hearing through animal experimentation.

The very exact and highly interesting experiments on acoustic responses in animals published by Wever and Bray[1] were shortly followed by Adrian,[2] by Hughson and Crowe,[3] by Davis and Saul,[4] and others. The majority of these experiments were done on decerebrated cats, and the electrical disturbances which were produced were led off from the auditory nerve or from the brain, and were in the forms of action currents.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication June 22, 1933.

*This research was aided by a grant from the Committee on Scientific Research of the American Medical Association and the Otologic Fund of the N. Y. Post-Graduate Medical School. I wish to express my thanks to Dr. Max Rabbiner for his valuable assistance in this work.

The writer's experiments of more than two years were also carried out on cats, but he did not decerebrate the animals and did not confine himself to leading off an action current from the auditory nerve.

Procedure: The writer experimented on normal cats, laid bare the bulla, then opened it and exposed the promontory or bony cochlea, and the round and oval windows. One copper electrode was applied to the bony cochlea and the other electrode to an indifferent part of the wound. Then both electrodes were connected to the input of a three stage amplifier. The output wires were led to earphones placed quite a distance outside of the operating room, out of earshot, where an assistant listened to the sound of a whistle, which was sounded near the ear of the animal and transmitted to the assistant through the amplifier. The assistant responded to the writer by a buzzer signal. When the active electrode was placed on an indifferent, outer part of the wound, the sound of the whistle blown by the operator was not heard by the assistant.

The severance of the clearly exposed auditory nerve, between the internal meatus and the medulla oblongata, did not interfere with the transmission of the sound. That the nerve was completely severed was proved by a subsequent autopsy on the animal.

When the secondary drum membrane of the round window was perforated and a physiological saline, or a camphor, or a strychnine solution was injected, the sound of the whistle was heard by the assistant. The sound was not heard, however, when a drop of quinine, salicyl, cocaine, or pilocarpine solution was injected into the round window, or when a minute lesion was made into the bony cochlea. Death of the animal also destroyed the electrical disturbance in the cochlea.

CONCLUSIONS.

A possible interpretation of the above enumerated experimental results may perhaps be found in the following suggestions:

1. The fact that a minute lesion in the bony cochlea (viz., clipping off a small piece of bone from the round window), or a minute perforation of the bony wall of the cochlea completely destroys the electrical potential in the cochlea is rather surprising. One could well understand why, in accordance with the Helmholtz theory, the stimuli of certain specific frequencies which were interfered with by the artificial lesion should not be transmitted to the assistant.

But why the total effect of all sound should be lost because of a minute lesion in the cochlea is difficult to understand.

2. The fact that certain chemical solutions mentioned above destroy the electrical potentials seems to suggest that these chemical solutions produce certain changes in the metabolic processes of certain cells, possibly in Corti's organ, which may be responsible for the origin of the electric disturbance.

3. The fact that the electrical potential produced by sound was not destroyed by the severance of the auditory nerve seems to suggest: *a.* that the cochlea, itself, is the source of the electrical potential; *b.* that the cochlea acts as a translating device, which converts the mechanical energy into an electrical one; *c.* that the electrical potential produced by sound in the cochlea acts upon the cochlear nerve as any electrical stimulus would act upon a motor nerve, and *d.* that the primary stimulus to the auditory nerve is electrical and not mechanical in nature.

An analogy between the action of the cochlea and a piezo microphone, in which mineral crystals produce electrical pulsations when acted upon by sound pressure, is suggested.

Subsequent investigation must determine what specific structure in the cochlea converts the mechanical air pressure constituting sound into an electrical potential.

REFERENCES.

1. WEVER, E. G., and BRAY, C. W.: The Nature of Acoustic Response. *Jour. Exper. Psychology*, Vol. 13, pp. 373-378, Oct. 5, 1930.

2. ADRIAN, E. D.: The Microphonic Action of the Cochlea. *Jour. Physiology*, Vol. 71, April 24, 1931.

3. HUGHSON, W., and CROWE, S. J.: Function of the Round Window, An Experimental Study. *Jour. A. M. A.*, Vol. 96, pp. 2027-2028, June, 1931.

4. DAVIS, J., and SAUL, L. J.: Action Currents in the Auditory Tracts of the Midbrain of the Cat. *Science*, Vol. 75, p. 105, Jan. 22, 1932.

1235 Park Ave.

THE HARD OF HEARING SCHOOL TEACHER.*

DR. HORACE NEWHART, Minneapolis, Minn.

"What per cent of hearing loss should disqualify a teacher for continuing her work?" This question is being asked the otologist with increasing frequency. The possibility of more accurately measuring hearing acuity by means of the audiometer, on first thought, would seem to make a satisfactory answer a simple matter. The increasing economic need in these times for clinging fast to a remunerative position gives added importance to the question. Its hasty or ill-advised answer may mean tragic consequences in the professional and economic security of many a worthy teacher.

When this question was first put to the writer by Dr. F. E. Harrington, Director of the Department of Hygiene of the Minneapolis Public Schools, with the expectation that a concise, direct answer would be readily forthcoming, we realized that the responsibility involved was not to be taken lightly. This responsibility assumed greater proportions when the same question was soon repeated by a number of anxious teachers. They were greatly perturbed by rumors that all teachers in the city schools were to be subjected to the same audiometric tests as are employed in checking the hearing of all the school children. We became very conscious of the far-reaching possibilities of injustice if such tests were made compulsory and the results to be used as the sole criterion of a teacher's right to hold her position.

Before attempting to formulate an answer that might carry the weight of authority we consulted by letter a number of men and women of national reputation in the fields of otology, education, speech correction, acoustic engineering and social service. The outstanding feature of their generous and helpful response was a striking unanimity of opinion, which with our personal views we have endeavored to summarize in the following paragraphs.

The problem of the hard of hearing school teacher is one of large importance. Compared with the problem of the hard of hear-

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication August 15, 1933.

*Read before Fourteenth Annual Meeting, American Federation of Organizations for Hard of Hearing, Chicago, June 19, 1933.

ing school child, now fairly well recognized in many enlightened communities, the problem of the hard of hearing school teacher has received very little attention. It affects with serious possibilities the professional careers and happiness of thousands of our most valuable citizens—our teachers. At a conservative estimate, at least four per cent of the 680,000 public school teachers in the United States, or between 25,000 and 30,000, have a hearing loss sufficient to impair, in varying degree, their efficiency in the class room. The problem is also one of real concern to the parent, the educational administrator and the taxpayer. But above all others it vitally affects the highest welfare of the school child.

With this recognition of the importance of the subject, it should be stated that it was emphatically the concensus of opinion of all the representative group who were consulted that no experienced or successful teacher can be fairly judged as to her fitness to carry on her professional work on the basis of a hearing test alone. It requires something vastly more comprehensive to disclose a teacher's capacity to successfully perform her professional duties. Other very important factors must be taken into account if one would justly evaluate the teacher's efficiency and worth. These factors may be collectively represented by her daily record of professional achievement which is distinctly a matter of individual, personal make-up. Each case, therefore, must be judged strictly on its own merits.

A teacher may have a hearing loss of twenty-five or thirty per cent and, exceptionally, even a materially greater defect and still do excellent work. This statement is conditioned upon such important factors as aptitude, a good educational background, experience, character, personality and interest in her work. More than all this, she must have readjusted herself by acquiring skill in the art of lip-reading and if her condition is such as to make it an advantage, she must supplement her diminished hearing power with a modern hearing aid. Such a teacher not infrequently achieves daily a far greater success as measured by results than many others possessing normal or only slightly impaired hearing, but who are lacking in some of the other necessary qualifications.

The arbitrary dismissal from the service of an experienced teacher of the former type because of a hearing loss would be extremely unjust and would deprive the school system of one rendering conspicuously fine service. Such action, however, can be taken and is taken in many instances, especially in communities in which there

is a ruling or a law that no teacher having a physical defect may be retained on the payroll. Personal prejudice, lack of understanding, political or economic pressure and other causes may tempt the school administrator to make an obvious or even a slight hearing loss the excuse for arbitrarily dismissing a successful, excellent teacher unless her position is safeguarded by friends who demand the recognition of her compensating qualities.

For the acquisition of correct speech the possession of practically normal hearing on the part of both the pupil and the teacher is a fundamental necessity. Accordingly it is self-evident that the teacher in the kindergarten and primary grades, whose most important duty is the detection and correction of faulty speech in her young charges, must be possessed of practically perfect hearing acuity. The same applies to teachers specializing in speech correction.

We must recognize a different situation in the case of teachers of the adult deaf. It has long been observed that among the most successful teachers of lip-reading for adults are many who themselves have a handicapping hearing loss. It is conceded that their victory over their own handicap gives them a better understanding of the needs of others similarly afflicted. Their interest, enthusiasm and courage, together with their fine example of attainment, inspire confidence and persistent effort on the part of their pupils. This results in a degree of success which might not be attained by some teacher with normal hearing, but not endowed with the same amount of zeal, interest and unselfish devotion as the hard of hearing teacher has acquired through her personal experience in overcoming her own handicap.

In the higher fields of education where older individuals in smaller groups are concerned, even a severe hearing defect plays a relatively small role in determining the fitness of the instructor.

The problem of the hard of hearing teacher cannot be readily solved until its importance is recognized. Indifference toward all matters pertaining to the conservation of hearing, the almost universal tendency to neglect ear diseases which cause deafness, and the common fallacy of trying to conceal a hearing defect have seriously delayed progress in reducing the number of hard of hearing individuals in all walks of life. There is great need of a wider dissemination of knowledge on this subject. This better understanding the hard of hearing people, largely through the organized efforts of the American Federation of Organizations for the Hard

of Hearing, are striving to accomplish with heroic courage and a fine optimism.

Those who have given this subject deliberate consideration would suggest the following recommendations as part of a program for solving the problem of the hard of hearing teacher.

A reduction in the incidence of a significant hearing loss and the salvaging for the greatest usefulness the hearing of those teachers who have a defect must be the first objective.

It is entirely fitting and a first essential of any plan that will yield success that every person applying for admission to a teachers college should be required to undergo a careful hearing test, adequate to disclose any slight hearing loss. The discovery of such a loss demands a thorough examination by a competent otologist whose opinion as to the fitness of the applicant to enter the teaching profession should be final. It is a kindness to advise such a person to seek some other occupation in which the possession of perfect hearing is less vital.

No candidate for a teaching position should be employed without a similar examination. It is far easier to reject an applicant with defective hearing than to dismiss one after she has been given a contract.

All teachers actively employed should be given a periodic hearing test as a protection against the insidious onset of a hearing loss of which she may not be aware. They should realize that voluntary submission to such a test is greatly to their advantage. In many cases an incipient ear condition may be revealed in time to permit of its correction, thus averting a serious hearing impairment.

The employed teacher who already has a loss of hearing, as an important duty to herself and to her pupils, should not attempt to conceal her defect. This is naturally her first impulse, fearing that the discovery of her impairment will cause her to lose her position. This is a serious mistake made by the majority of hard of hearing persons. The teacher who attempts to conceal her handicap, inevitably, in time loses the confidence of her pupils and is often the object of ridicule. Were she to acknowledge her defect she would win the co-operation of her pupils and be more likely to receive the understanding and help of her superior and principal. An early readjustment of her work, such as a transfer to a position dealing with older groups, or to some other department of the school sys-

tem in which a diminished hearing acuity would be no disadvantage, often could be arranged and dismissal avoided.

A teacher having discovered that she has a hearing loss should first of all make every effort, with the help of the best medical skill she can command, to discover and remove the causes in the hope of restoring her hearing or at least preventing its further deterioration. If her loss is already sufficient to constitute a handicap, she should lose no time in trying to preserve what hearing power she still possesses by such means as a conscientious physician may advise. This often means the adoption of a new health program. Local treatment should be persisted in so far as it may improve her condition or retard the progress of the underlying causes of her hearing loss. She must be warned to avoid poor medical advice and especially pseudo-medical advice or quackery. The preservation of even a small amount of residual hearing in these days of greatly improved hearing aids is most important. She should at once begin to perfect herself in the art of lip-reading to compensate for her diminished hearing acuity. When her condition is such that she can advantageously use a hearing aid, she should waive all pride and prejudice and secure at an early date the type of instrument best adapted to her need and use it with no more self-consciousness or embarrassment than the average person uses glasses to compensate for a defect of vision. By thus rehabilitating herself, she can for years successfully carry on instead of admitting her defeat and giving up in disappointment and sorrow her chosen life work.

The average conscientious teacher as soon as she knows her hearing loss seriously affects her work will generously resign her position, knowing that in ordinary times of prosperity, with her larger intelligence and better adaptability, she can make a place for herself in the complex struggle for existence.

It is the privilege of the otologist and the administrator of our public school systems to consistently and fairly lend their best cooperation to avoid the arbitrary dismissal of any worthy hard of hearing teacher without an impartial study of all the facts in each individual case, always mindful, however, of the best interests of the pupil.

527 Medical Arts Building.

LEPROSY OF THE EYE, EAR, NOSE AND THROAT.*

DR. F. J. PINKERTON, Honolulu, Hawaii.

This paper is presented as a brief review of observations made in the past twelve years of investigation and study of the disease. As we recall the many cases that have passed through our hands and correlate our findings from our experience, we regret that we have so little to offer in the way of a cure or remedy for so many leprous lesions of the eye, ear, nose, throat and larynx.

Of one thing, however, we are sure, that leprosy rarely, if ever, exists in a patient who does not also suffer some lesion of the eye, ear, nose, throat or larynx at some time during the course of the disease, in most cases the lesions being disabling in some degree, in many cases destroying sight, frequently interfering with the physiological function of the nose, or producing serious impairment of the laryngeal mechanism.

In my report on "Leprosy of the Eye," in 1927, I concluded that, "Any patient suffering from leprosy will probably have, sooner or later, some involvement of the eye." By this statement I meant every case with progressive, uncured or unarrested leprosy; that is, those cases that never recover from the disease. At this time I have experienced no change of opinion and I believe that the above is not an exaggerated statement. I further feel that most, by far the great majority of patients with progressive leprosy, uncured and unarrested, will also have some lesion involving the ear, nose, throat or larynx.

For some reason, the eye seems to be more certain of involvement, though our surveys of nose, throat and ear conditions have not been as extensive as our eye surveys, nor have we had the cases under observation as long as we have had the eye cases.

The opinion expressed in 1927 regarding the eye remains the same so far as treatment is concerned with the exception of some modifications of technique which offer some improvement in the

*Read before the Hawaii Territorial Medical Association, April 29, 1933.
Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 15, 1933.

cosmetic result in the lid plastic work and also less formidable procedures in treating the limbus nodules.

Lid Paralysis: When we first inspect a leprosy case, we instinctively observe the facial appearance, noting nodules, swellings or edema, macules, absence of brows, and lashes, paralysis, etc. Frequently we see nothing but a sagging lower lid, a tremor, or an absence of one or both nasolabial folds, which latter is characteristic of a facial paralysis. When the patient attempts to close his eyes, the diagnosis of orbicular paralysis or more complete facial paralysis is often easily made and in doubtful cases can be confirmed by using the fingers to resist the effort of eye closing, thus frequently detecting a weakness in one or both orbicular muscles. The question now arises, Does this case, because of his facial nerve lesion, require any treatment?

I have proceeded along two lines of treatment. First, conservative; second, radical. In the conservative management, *1.* we attempt to teach the patient what the condition is and how best to conserve his eyesight, inasmuch as this condition is practically always permanent. We routinely advise daily cleansing treatment and the use of a mild antiseptic ointment at night, or the wearing of a patch to keep the lids closed and the cornea covered. In patients of low mentality or a poor co-operative attitude, we advise and urge them to permit the more radical surgical procedure, *2.* which is that of a tarsorrhaphy operation at the external canthus.

While a lid palsy or orbicular paralysis is not painful *per se,* I have observed that neglected lid conditions are very frequently followed by corneal changes which are directly or indirectly due to corneal exposure. Ophthalmologists are familiar with a definite corneal lesion, Keratitis E Lagophthalmo that is mentioned in all textbooks. There is also described in most textbooks a surgical method for correcting the lid deformity. Perhaps one of the most well known lid operations is that of Fuchs. My first hundred or so attempts were carried out strictly along the lines of Fuchs' technique. I was disappointed in the cosmetic results of the Fuchs operation, the patients complaining of the "pig eye" effect that resulted. So, in consultation with Fuchs, I modified his technique by carrying my external incision higher up and more temporalward, anchoring the lower lid to this new site. The disadvantage of this procedure was to create a new vertical fold or wrinkle of the skin at the temporal margin of the orbit, unsatisfactory from a cosmetic standpoint. So, more recently, I have overcome this difficulty by excising a tri-

angular section of skin, at the temporal margin of the orbit, thus removing the excess of flaccid skin and putting the lower lid on the stretch, at the same time actually elevating or lifting the temporal extremity or anchorage of the lower lid. This modified technique very satisfactorily corrects the deformity and gives a good cosmetic result. Patients do not object to this new technique as they did to the old.

This modified technique which I have described above is the result of trial and error of various other procedures. In viewing the literature on the surgical repair of leprous lid paralysis, we find no reference made to the surgical repair of those lid conditions, consequently we have had to devise new measures to fit new conditions especially common to leprosy. One cosmetic defect of this modified technique is the elongation of the lower eyelash line temporalward, which is of no great importance in a large number of patients who have already lost their eyelashes or whose lashes are thin and sparse, due to leprosy; but in those cases with good eyelashes this lower lid transposition is distinctly disadvantageous. This can in most cases be reduced to a minimum by excising the hair follicles with a sharp scissors.

My contention has always been, that in these complete orbicular paralysis cases, we should not wait until corneal changes have taken place. We know from experience that corneal changes will almost inevitably occur, and we urge and advise these patients to have some lid plastic work done early, rather than late.

Lacrimal Gland: I have nothing new to offer in the medical or surgical treatment of the hypertrophied lacrimal gland. Since it frequently becomes enlarged secondarily to lid paralysis, it usually subsides when the proper lid operation has been done. In the few operations that I have done, only a portion of the accessory lacrimal gland is excised after the technique of De Wecker.

Lacrimal Sac: In my experience there is no disease of the lacrimal sac or duct peculiar to leprosy other than the secondary involvement from leprous lesions of the nose, which is a rather common complication. It usually promptly improves when the nose is adequately cleaned out and hygiene to the conjuctival sac is instituted.

The Conjunctiva: The conjunctiva of a leprosy patient is subject to the same infections and diseases as other patients, plus those lesions that are primarily or secondarily due to leprosy. Naturally, in all lid palsies the conjunctiva is in a constant state of irritation

and it is commonly reddened and thickened by mechanical irritation. In the lid palsy condition, we frequently see pinguecula and pterygial types of thickenings, which are usually treated surgically along with the lid operations, if they are of such size as to warrant removal.

It is common to observe leprous nodules or granulomas of the conjunctiva usually in the region of the temporal limbus, though they may occur in any area of the limbus. The nodules may spring from the conjunctiva itself or beneath the conjunctiva, pushing it forward as a rounded firm nodular mass adherent to the sclera or springing from the sclera or through the sclera from the ciliary body. These are best treated by surgical dissection or electrocoagulation.

The Cornea: The cornea is frequently the site of a leprous lesion either as a primary lesion or secondarily from a lesion at the limbus encroaching through the corneal layers. The cornea is commonly steamy due to erosion from traumatism of dust and dirt and constant exposure, or from the drying process due to lack of lid movement to keep it moistened. This keratitis from exposure is best treated by lid repairs, frequent cleansing and some mild ointment during sleep.

Nodules at the Limbus: Deep nodules at the limbus usually spring from the ciliary body, gradually growing forward through the sclera until they produce a rounded mass in the form of a fixed nodule. At this juncture they may break down into a granulomatous mass, gradually involving more and more of the cornea until they project through the palpebral fissure as a lepromatous granuloma. They are best treated by surgical removal, cauterization or, preferably, electrocoagulation. The use of electrocoagulation offers a more certain destruction of the mass with less danger to the important ciliary zone below and when properly applied, even when the mass springs from the ciliary body, they are more surely and completely destroyed by the electrocoagulation method than by surgical removal. If treated early much can be done to stop their progress and prolong useful vision.

Extrinsic Ocular Muscles: Considering the frequent incidence of other nerve lesions peculiar to leprosy, I have watched carefully for the development of some extra ocular muscle lesion and have found none that were not logically the result of other causes.

Fourth and Sixth Nerve: The fourth and sixth nerve in my experience have never been involved. I have never seen an involve-

ment of the third nerve except in very advanced and late cases where an extensive involvement of the iris and ciliary body were present. In such a case it is easy to understand that the damage was done to the terminal distribution of the nerve in the iris but that the nerve trunk proper was not involved.

The Iris: The iris is probably more frequently affected by leprosy than any other single structure of the eye or its adnexa. An attack of iritis does two essential damaging things to an eye,—the production of an exudate with pigment changes or pigment shifting plus an atrophy of the iris structure. While each attack may run its course without treatment, there is the great likelihood that even a single attack, unattended, will produce such extensive damage to the iris that vision is seriously impaired and such changes result as to interfere with good results from subsequent treatment, no matter how efficiently it may be carried out.

So, it is essential that prompt and early atropinization be carried out and its use is recommended and taught in every early case of iritis, as well as later. By its use we prevent the formation of synechiae or adhesions of the iris to the anterior lens capsule, and the iris is thus put to rest to conserve its muscular and nerve structures. It is a common experience to observe that the iris shows absolutely no response to atropin after an attack or two of inflammation due to leprosy. The use of hot compresses and dionin seem to be gratefully received by the patient, and their use appears to be a helpful adjunct to the more important use of atropin.

Three schools of thought seem to exist about how leprosy finds its way into apparently normal tissue. A. Pillat, of Vienna, who has had experience with leprosy in China and who is a real student and a tireless worker, interested in investigative studies, believes in part, that leprous lesions of the eye occur by tissue continuity and by the organism being implanted on the surface of the part or parts affected. Thus, it would appear that leprous adhesions occur by simple contact of an active organism to a vulnerable spot or location on the skin or mucous membrane. It reaches deeper structures by further penetration due to possible motility of the bacterium.

Fuchs considered the possibility of the organism having the power of locomotion or motility. This idea resulted from his studies of the corneal lesions, wherein the organism motivates its way into the interlamellar spaces of the corneal stroma proper, in which no apparent disease was present.

I concur in both Pillat's and Fuchs' ideas but I would also add a third thought; by blood and lymph transmission. We so frequently note that structures commonly affected also have a good blood supply, and while the organisms are found in locations of scanty blood supply, we do not find rapidly growing nodules in such parts as we do in parts of good blood supply and especially those parts that are subjected to injury or excessive motility.

Intraocular Media: The anterior chamber remains clear until the ciliary body becomes involved, when there is exhibited the usual phenomena that occurs in cyclitis of other causes. Deposits on the posterior corneal surface are common and hypopyon is frequently to be seen where an advanced nodular process involves the ciliary and the root of the iris.

The lens appears not to be involved because of leprosy, until advanced changes occur in the ciliary body. When this is the case the lens is rarely seen easily enough to determine just what is taking place. I should think, however, that we might well say that the lens becomes cataractous in the later stages of a leprous ciliary disease.

The vitreous body in leprosy remains clear until advanced corneal, iris or ciliary changes make it impossible to see and study it clearly. Even though the vitreous cannot be seen, I am quite certain that it shares in the process of the ciliary involvement.

Fundus: I have examined the fundi of hundreds of leprous patients and I have never seen a case that I considered significant of leprosy. I have examined these eyes, always searching for nodules in the choroid, or an inflammation or atrophy of the optic nerve, with negative results. In the lesions that I have found I have felt justified in ascribing them to other and more positive causes.

The Nose: The external nose, especially the ala, is a common site for leprous nodules to occur. The alar regions and the upper lip seem to share in the acute edemas of leprosy and while the intranasal structures may share in the acute process by tissue continuity, it is not the rule.

Some of you may recall the statement made by Dr. Gorman, U.S.P.H.S., who was on duty at Kalihi Hospital in 1924. He called attention to "nosepicking," as a cause or contributing factor in leprous lesions of the nose. He considered the possibility of the patient carrying the organism by his fingers to the nose and implant-

ing it there, the "nose picking" procedure providing an additional irritation, thus favoring a proper culture media for a successful leprous growth.

The nasal mucous membrane undergoes the same general picture as occurs in the various manifestations of generalized leprosy, namely, invasion, edema, nodulation, ulceration and finally cicatrization. Unfortunately for the comfort of the patient, the thickening and fibrosis is often too extensive as to entirely disappear during the end-process or scar formation, with the result that these patients often continue on through life with more or less narrowing and atresia of one or both nares.

As stated in my article last year, the favorite site of a localized lesions in the nose is just posterior to the mucocutaneous area on the septum and the anterior extremity of the inferior turbinate. Eventually, or soon after these two points are infected, the remainder of the nasal mucosa becomes involved. In the ulcerative stage 8.5 per cent of the cases show a perforation of the cartilaginous septum.

The Pharynx: It is common to observe that lesions appear in the soft palate and pharynx some time subsequent to the nasal lesions and while I have not definitely concluded that the pharyngeal involvement always follows the nasal lesion, my experience leads me to this impression.

The base of the uvula or the area slightly lateralward to the uvula near the free margin of the soft palate is most frequently first involved, the nodular process often extending to the tonsil and including it.

The ulcerative stage is usually severe and we frequently find the uvula to be entirely destroyed and a similar destruction of the tonsils, though not as complete as the uvula. I have seen no instance of a perforation of the hard or soft palate, though I have wondered why such a perforation has not occurred, considering the lesions that occur in other parts, such as the foot, where deep ulcerations occur involving bone. The nodules may also extend forward and include the remainder of the hard and soft palate, also extending farther and farther to include the alveolar areas, both buccal and gingival. Two and one-half per cent of our cases show nodules or lesions of the tongue.

Our treatment of nasal and pharyngeal lesions has been quite varied. We have tried the recommendations of others, and have

instituted some of our own in an attempt to find something that would relieve the patient symptomatically. The various nasal packs have been tried, but our feeling is that our beneficial results have all been because of the cleansing of the parts and general attention to the patient as a whole. We can report no great improvement by local treatment alone, other than temporary gratification of the patient and also the psychic reaction that something is being done. In the pharyngeal lesions, excision of the nodules or electrocoagulation offers the best treatment thus far in our hands.

The Larynx: The larynx is often involved. Here again we are confronted with a picture which to the untrained observer very closely simulates that of tuberculosis. Experience of several years in the handling of a large number of tubercular laryngeal complications helps us greatly in making this differential diagnosis.

The details of differential diagnosis cannot be set forth adequately. One must form clinical impressions, that are perfectly clear to his eye but which are impossible to describe to another. And these clinical impressions are possible only after examining over and over again, case after case of both tuberculosis and leprosy of the larynx.

Experience teaches me that the same acute phenomena occur in the larynx as in other parts of the body, and it is easily understood that an inexperienced observer may be led astray in mistaking an acute generalized edema of the larynx for a chronic nodular thickening. The appearance is much the same, but one can make a distinction by constant study and observation. In doubtful cases it is well to keep the patient under observation for two or three weeks. Perhaps on the next examination a few days later, one will find the edema almost entirely gone and the larynx returned to a more normal condition. It would be a mistake to cauterize such a larynx when seen in this acute edematous condition.

The Epiglottis: The epiglottis is first seen and we note the striking characteristic of a pale infiltrated edematous mass during the early acute stage, later to become nodular and irregular. When it finally heals, after ulceration, we note its marked fixity, irregularity, destruction or distortion. Eighteen per cent of our cases showed involvement of the epiglottis. The aryepiglottic folds take part in the process in a similar manner, and the process extends then to the arytenoids. Here we find the pale infiltration process as at first, later to become irregular and nodular. The same is true with the ventricular bands and the voice becomes coarse and raspy because

of the tendency of the ventricular bands to overhang the true cords, plus the limitation of movement of the arytenoids. I have seen but two cases which I considered to be leprous nodules of the true vocal cords and while the true cords become edematous during the acute invasive tissue reaction of the contiguous structures, it is probable that nodules of the true cords are rather rare.

Profiting by our experience in treating other parts, we have resorted at once to the electrocautery treatment of the larynx as a routine. The best time for its application has been found to be during the nodular stage, and before ulceration begins. After cauterization we have noted marked reduction in the size of the nodule, thus affording the patient more space to breathe and bringing about an earlier scar formation than would otherwise be the case were repair left to Nature. Thus the tendency for an overgrowth of nodular tissue is curtailed, reducing the probability of doing a tracheotomy. Of course, such a procedure must be done early and its need recognized long before it becomes immediately necessary, as cauterization not infrequently produces some acute edema, which in turn would result in complete closure of the glottis already narrowed by disease. We have found that the use of the white heat cautery point is preferable to the cherry red heat, because the white hot tip penetrates quickly and does not cook the tissue remote from the punctured area. Also, by using the white hot wire there is less likelihood of the tissue becoming adherent to the cautery point and the resulting burn is not as painful as when a cherry red tip is used.

External Ear: As regards the external ear and its leprous affections, we are confronted with the same problems as involves the skin elsewhere in the body. While I have done but a limited amount of ear work in leprosy, I am convinced that CO_2 snow, electrocoagulation and the injection cutaneously of a strong counter irritant are the methods of choice. Of course the excision of a nodule may always be done with good results.

I have never seen a nodule of the external auditory canal, nor any other lesion that might be charged to leprosy. Most of the patients have a dry scaly dermatitis, and excessive cerumen seems to be a common condition.

Middle Ear: I have not seen a case of otitis media or lesion of the middle ear that might be considered as due to leprosy. Nor have I seen a case of deafness or reduced hearing that I felt was directly

the result of leprosy. While we have seen several cases of catarrhal or adhesive deafness, it is believed these might have been indirectly due to leprosy since there is such a common nasopharyngeal involvement in leprosy.

Internal Ear: I have not seen a case of nerve deafness in leprous patients that I felt was due to the disease, the only case of pure nerve deafness being in a patient with known syphilis of long standing. The vestibular apparatus seems to entirely escape involvement in leprosy.

Finally, while I have considered here only the subject of eye, ear, nose and throat, I am not unmindful of the great importance of the subject as a whole.

We have made definite progress in our leprosy program, increasingly so by the kindly and co-operative efforts of the medical profession of Hawaii, because most of you have become "leprosy conscious" and anxious to give a hand in this, our joint interest. The work of the U.S.P.H.S. Staff at Kalihi Hospital has contributed greatly to the spread of knowledge concerning leprosy among the physicians.

It has not been, nor will it ever be, a "one man job" even if he be a person who has had many years of experience and fundamental training in this interesting disease. I have at this time the duty and pleasure of expressing my deep appreciation to those of you who have constructively criticized and advised me on so many and varied angles of the work that constantly come up for consideration, and especially do I wish to express my appreciation and thanks to Dr. N. E. Wayson for his kindly efforts and advice in the many problems that I have talked over with him.

REFERENCES.

1. PINKERTON, F. J.: Leprosy of the Ear, Nose and Throat. *Archiv. Otolaryngol.*, pp. 469-487, Oct., 1932.

2. PINKERTON, F. J.: Surgery of the Leprous Eye. *Proc. First Pan-Pacific Surg. Conf.*, Honolulu, Hawaii, Aug. 14-24, 1929.

3. PINKERTON, F. J.: Histopathology of the Leprous Eye. *Trans. Hawaii Terr. Med. Assn.*, Honolulu, Hawaii, April, 1928.

4. PINKERTON, F. J.: Leprosy of the Eye. *Archiv. Ophthalmol.*, Vol. 56, No. 1, 1927.

DISCUSSION.

Dr. N. E. Wayson: I think that Dr. Pinkerton's contribution is very timely. He has invited your attention to the fact that leprosy is not exclusively a disease of the skin, but like syphilis, affects portions of the eyes,

nose and throat. In this community an iritis, episcleritis or a chronic proliferative rhinitis should be explored for the probability of a leprous origin. I believe it is most important that we rapidly change our conception of leprosy, and instead of thinking of it as a disease of foul-smelling sores and grotesque facial mutilations, we shall consider it as a communicable affection of protean manifestations, whose commencement may be acute or is so insidious many times that neither the patient nor his physician is aware of the presence of the infection.

Dr. Pinkerton has indicated that the structures of the nose and throat may take part in the acute manifestations of the disease. He has recognized that the clinical picture of leprosy may be quite as acute and fulminating as that of scarlet fever and of measles, and it should be known that at such times the membranes of the nose and throat usually take part in the reaction. It is even more important, however, that we should appreciate that these acute and subacute exacerbations or progressions are often indicated by changes in the eye well in advance of the appearance of the generally disseminated lesions; that is, a keratitis, iritis or iridocyclitis (or a persistent "cold" in the nose) may be the prodromal sign of widespread changes which may continue slowly or rapidly.

Many of the changes described by Dr. Pinkerton are those observed in cases of moderate or marked advancement, and which are accompanied by destruction of tissues with scarring and contractures. I would like to emphasize a fact which I think he is aware of; namely, that many paralyses which result in lagophthalmos are not followed by conjunctival damage through exposure of the eyeball in the course of several or many years. Also, it should be made clear that in many cases of paralysis of the lower lid there is a recovery to such degree that the individual has good, if not perfect function, and surgical treatment should not be resorted to until the degree of regeneration of the nerve, or of spontaneous restoration of function has been definitely determined. Dr. Pinkerton has indicated that such is the case in the treatment of laryngeal and nasal affections, but it does not seem to me that he has sufficiently emphasized this point with regard to involvement of the eye. On the other hand, an acute leprous inflammation of the eye is sometimes accompanied by very rapid and extensive ulcerations in the conjunctiva, whose treatment appears to be greatly facilitated by a partial tarsorraphy, just as one finds in the neurokeratitis resulting from affections of the ophthalmic branch of the Vth nerve. There appear to be factors of disturbed nutrition of the tissues at work, rather than purely mechanical irritation.

It should also be evident that when one is considering these eye, nose or throat lesions of the moderately advanced or advanced case, there will be other clinical manifestations which contribute to the diagnosis. However, the conditions in the eye, nose or throat may be those which bring the patient to the physician and their recognition will stimulate him to more extensive examination in confirmation of his diagnosis.

Again it seems reasonable to believe that a more detailed study of the eye, nose and throat affections may result in the earlier recognition of cases and contribute to a better control of leprosy from the public health standpoint. This is especially true if microscopic examination of preparations from the nasal mucous membrane is made.

A CASE OF DOUBLE BRAIN ABSCESS.

Dr. H. D. Newkirk, Anaheim, Cal.

The following case is reported for several reasons: *1.* The seeming lack of etiologic history. *2.* The large abscess masses possible within the brain substance for a long period of time with no clinical symptoms. *3.* The surgical technique with complete enucleation of abscess in capsule.

The pathologic report appended is by Dr. Cyril B. Courville, resident neuropathologist at the Los Angeles General Hospital.

The patient, Miss E. V., age 14 years, a brilliant first-year high school student, was first seen by me, Nov. 17, 1932. Her chief complaint was headache, severe and progressive.

Past History: Her parents stated that her health had been good until two months ago. She had had the usual childhood diseases with apparent full recovery. She has not matured yet. There was no history of discharging ears or sinus infection, although she had occasional colds with no other than the usual symptoms of acute rhinitis. There was no chronic purulent nasal discharge.

Present Illness: The present illness began two months ago with mild headache increasing in severity. Her mother does not recall any acute febrile illness at that time. She was taken to two medical doctors and one chiropractor without relief. The main location of the pain on date of entry was the right frontoparietal region and was worse at night. She was able to walk in a straight line, eyes open or shut. She had no nystagmus, but vertigo on arising. She had had diplopia for the past two weeks. There was marked weakness with loss of weight from 100 pounds to 89 pounds, and she had a very sallow complexion. Some constipation was present. For the past week, there had been occasional vomiting of the projectile type.

Examination showed a tall, slender, sallow complected girl. General physical of chest, heart, lungs and abdomen was negative. Temperature was normal and pulse 65. Urine was negative. Blood: Hbg., 87 per cent; R.B.C., 3,930,000; W.B.C., 10,600; polys., 71

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 15, 1933.

per cent; small lymphs., 7 per cent; large lymphs., 7 per cent; trans., 3 per cent; baso., 1 per cent. No change in size or shape of red cells.

Neurologic Examination: All cranial nerves normal except paralysis of right external rectus. Pupils equal and reacted normally. Right disc showed papillitis of five diopters; left, three diopters. Vision right = 20/200, corrects to 20/100 and Jaeger number 10. Left = 20/20 and J. number 1. Unable to co-operate for fields.

Spinal puncture was done with great caution. Fifty cc. of clear fluid was taken; original pressure, 24 mm. Hg. Laboratory examination of this fluid was: Cell count, five lymphocytes; globulin, slight trace; and Wassermann negative. Fifty cc. of air was reinjected.

A series of encephalograms were taken (lying down), which showed a large oval mass, encapsulated in right frontoparietal region. This was thought to be a tumor.

The patient was at once sent to the hospital for a short observation. During the next two or three days, she failed rapidly. Her pulse rate continued to drop, vision to blur, and vomiting and headache to increase. She was soon too lethargic to answer questions.

Operative Procedure on First Entry: Under avertin anesthesia, a fairly large bone flap was turned down over the right frontoparietal region. Also a fairly large decompression area was rongeured out anteriorly. The dura was examined, but nothing abnormal was noted except the increased intracranial pressure. A large dural flap was then opened from below up. There was no adhesion of the dura to the cortex. The cortex seemed quite normal except for a possible slight discoloration in the area under suspicion. An Adson trocar was inserted in the suspected region and at a depth of about 5 to 7 mm. thick pus was found. The opening was enlarged a little and two rubber tube drains inserted and well fastened. Rapid improvement in general condition followed, and she left the hospital three weeks from date of operation. There was still free pus discharge and some temperature, but she was eating and sleeping well. Headache, vertigo and vomiting had ceased.

Second Entry: Two weeks later the temperature began to rise again and patient was hopitalized for observation. She was very irritable, but quite rational and had few symptoms. She vomited once. A rather large soft hernia was evident in the area of decom-

pression. This was opened and about a half ounce of clear fluid poured out. The temperature and pulse continued to rise and irritability with some illusions was present. There was no marked pain.

On Jan. 6, with no anesthetic except morphine sulphate, grs. 1/4 and atropin grs. 1/150, dissection was made about the area of the drain, one of which was still in about one inch deep. By finger

Fig. 1. One of series of encephalograms showing tumor mass. 50 cc. spinal fluid withdrawn and replaced with air.

dissection, the stalk or capsule of the abscess was followed in to the center of the frontal lobe, where the large mass showing in the X-ray could be plainly felt. The whole mass was carefully enucleated and it seemed to have no bony attachments whatever. Moderate bleeding was stopped by gauze packing, and the patient returned to bed, seemingly having suffered very little shock. Subsequent days were very stormy, the pulse especially remaining con-

tinuously high. Patient was semidelirious much of the time. Occasional vomiting, and involuntary actions of both bladder and rectum were present, and there was some weakness of left side of face, left arm and leg.

Slow but gradual improvement took place as the packing was decreased. The paresis of face, arm and leg returned to normal and paralysis of right rectus also entirely cleared up. The papillitis has receded and vision now is: right, 20/20, and left, 20/20. Mentality is entirely normal and patient has gained about twenty-five pounds in weight.

Examination of the specimen shows an elongated mass, the inner part being hard and almost circular. This seemed attached to, but not directly connected with the outer shrunken mass, which was the outer abscess drained on first entry. There were probably two adjoining abscesses. There were no particles of bone attached to either abscess. The thickness of the capsule is very interesting. It seems hardly possible that such a thick (one-quarter inch in places) fibrous capsule could have formed in two months. The pus was a pure culture of staphylococcus.

The thickness of the walls of the two abscesses was not known at the time of first operation, nor was it known that there were two separate masses. While the wall of the inner abscess proved to be very thick, the wall of the outer abscess, near the cortex, was thin and necrotic. The blunt trocar went through almost by its own weight; thus, it will be seen that it would have been impossible to enucleate the outer abscess even though one had intended to do this from the start. At the time of draining the first abscess, it was thought that there was only one and that eventually this would drain by the usual method. The writer realizes that it would be very poor surgery to intentionally do as was done, namely, drain one abscess and then through an infected area, digitally remove a mass deep within the brain substance; however, the surgery which was done was not deliberately planned that way. It was only after the first operation was finished and the fact of two separate masses established, that it became quite apparent something further would have to be done through this infected area. Under the circumstances then, when on further exploration the second mass was encountered, and finding that there was no pathway from the first abscess to the second and finding, too, that probing the second mass merely pushed it a little bit rather than entered it, was the procedure followed as described. It was still thought that the inner mass was a

tumor even though the outer one had proved itself to be an actual abscess.

GROSS PATHOLOGY.

The specimen which was brought into the laboratory by Dr. Harris D. Newkirk consisted of a brain abscess capsule to which was attached a heavy fibrous connective tissue stalk. The abscess cavity measured 4x3x3 cm. in its greatest diameters and the irregular stalk measured about 5 cm. over all in length. The main capsule was irregularly egg-shaped. Still adherent to its outer surface was some degenerated brain tissue. This gave the capsule a rather ragged and irregular appearance. The stalk was somewhat irregular in shape, being larger adjacent to the abscess capsule and then becoming progressively smaller as it approached its termination lodged in the cortex of the brain.

When the abscess was opened it was found to be markedly thickened, varying from 3 to 5 or 6 mm., depending on the situation. It presented a definite whitish striation, characteristic of old connective tissue. It was lined by an irregular pyogenic membrane. The contained pus was a grayish-yellow color and was rather thick and tenacious.

The abscess was said to have been removed from the anterior and middle portion of the right frontal lobe, the encapsulated portion having been recovered at the second operation by following down the tract of the stalk. It was removed digitally.

HISTOPATHOLOGY.

Section I—Hematoxylin and Eosin: The abscess wall consists of four layers, as shown in the routine H & E stain. The innermost layer was composed of polymorphonuclear wandering cells embedded in a gelatinous brown substance.

Under higher magnifications the appearance and arrangement of the coagulum makes it evident that it is the result of a degeneration of wandering cells which formerly constituted the exudate in the abscess cavity. The remaining wandering cells consists of multinucleated leukocytes and mononuclears with irregular nuclear outlines. Many of these cells are undergoing acute degenerative changes.

The second layer, lying just within the layer of exudate and from which it is fairly sharply demarcated, consists of numerous wandering cells in an excellent state of preservation, together with

numerous fat granular cells and fibroblasts. The wandering cells are largely polymorphonuclear and mononuclear. Plasma cells were almost entirely absent except in the more cellular foci found near the deeper portions of this layer. This layer has a finely granular appearance which, under higher magnification, was found to be due to the occurrence of numerous fat granular cells. A few multinucleated giant cells were observed in this layer, and in its deeper portions new formed blood vessels could be found extending in diminishing size and numbers to its peripheral margin.

The third layer, or capsule proper, consists of closely packed connective tissue fibres which have undergone some hyaline change. In this connective tissue layer, particularly along its inner border, are

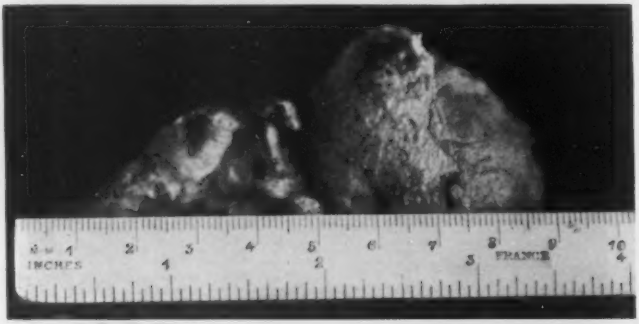


Fig. 2. Both abscesses. Mass on left is first abscess (capsule only). Drained on first entry and attached to second abscess still intact.

found collections of wandering cells, including many plasma cells. These fibre cells are found throughout the layer as still being numerous in its outer portion, although more diffusely scattered.

This connective tissue layer is not entirely homogenous and in fact in no situation of the section of capsule under study was it completely solid. The general tendency is to have a narrow zone in which the connective tissue fibres form a network merging gradually with the underlying layer. Then it becomes more solid, of varying width. This layer is interrupted at intervals by large collections of wandering cells which seem to have broken through it. There is again an outermost, more solid layer separated from the inner one by a more loosely reticular tissue in which wandering cells are diffusely scattered. Therefore, the connective tissue layer consists of

two fairly solid layers with an intervening reticular layer in which wandering cells are found in large numbers. The layer again fades out into the overlying brain substance in some situations, although for the most part the outer margin is more sharply delimited than the inner.

The outermost layer is composed of degenerating brain tissue in which all evidence of any structure is lost, having either a coagulated or reticular appearance. The nuclei of the glial cells are large and some of the cells are multinucleated. There does not seem to

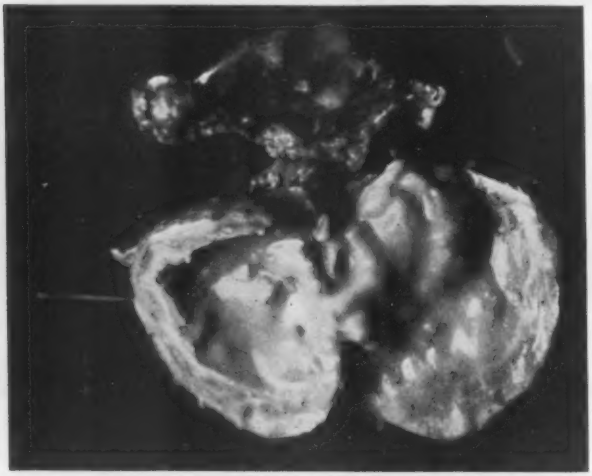


Fig. 3. Second abscess opened, showing thickness of capsule. Mass below is shrunken capsule of first abscess. Pus was pure culture of staphylococcus.

be a marked local gliosis. Some of the adjacent astrocytes have undergone hyalin degeneration. Collections of wandering cells, chiefly mononuclears, are found about the blood vessels in this area.

Section II—Scharlach R for Fat: The first internal layer of the exudate is marked by fine fat globules within the degenerating leukocytes.

In the second layer the fat is included for the most part within well defined round, oval or slightly irregular phagocytes. In many places this layer is sharply marked off from the exudate layer by a more or less solid row of fat granular cells. These fat granular

cells again disappear quite suddenly at the outer margin of the collections of wandering cells which border the underlying connective tissue layer, although a few such cells are found within such collections. In the deeper solid portion of the connective tissue membrane there are practically no fat granular cells, while in the loose reticular layer between the two solid portions are found large collections of phagocytes. They are also found scattered throughout the outer connective tissue layer, many times in the form of elongated cells, particularly in the region of the blood vessels. They are found in small numbers, and usually also of small size, in the brain tissue outside the capsule.

Section III—Reticulum Stain: The layer of exudate appears as a diffuse granular layer, sharply marked off from the next layer by a lacelike border. This second layer has a finely reticulated or lacelike appearance, due to the collection of fibres about smaller open spaces, evidently occupied by the fat granular cells. If this is true it is evident that these cells can persist for a long time, long enough for connective tissue fibres to form a meshwork about them. This layer has a slate-gray appearance, being lighter in color than the underlying connective tissue layer. The arrangements of fibres vary somewhat, occurring in the form of whorls about the blood vessels or assuming a radiating arrangement toward the center of the abscess cavity. This layer is found to vary considerably in thickness.

The underlying connective tissue layer is composed of dense fibre connective tissue strands, forming the outer and inner membranes of the capsule with looser strands lying between the two.

In the degenerating brain in the external layer are found a decreasing number of blood vessels which assume a parallel arrangement for the most part between which a reticular connective tissue forms a complex network. The width of this layer varies considerably, being wide where numerous wandering cells and phagocytes occur, and narrow where these are absent.

Diagnosis: Brain abscess, possibly rhinogenic, obviously of some age.

Johnston Clinic.

INTRACRANIAL COMPLICATIONS APPEARING DURING THE TREATMENT OF EAR AND NOSE DISEASES.

DR. ERNEST REEVES, Passaic, N. J.

Nothing is so irritating to the specialist as the sudden development of signs of intracranial complications following treatment of ear suppurations or acute sinus disease. His irritation is well grounded as these complications are very dangerous, with frequent fatal termination.

In addition to that, they are not at all common and their diagnosis and treatment is a very difficult problem not only for the oto-rhino-laryngologist but even for the whole expert staff of a specializing institution.

An incorrect diagnosis of a brain abscess may cause one to open the dura and probe the brain, producing accidental meningitis or encephalitis; but a delay in brain abscess may result in rupture, meningitis and death.

In presenting the following five cases, I will attempt to clear up some difficulties encountered in the diagnosis and treatment of such intracranial complications.

Case 1: Thirty-eight-year-old male. When I first saw him, he appeared very ill, semicomatose. Following history was obtained from his family: Discharging ears for two years. Hearing very bad. In past year had several attacks of stomach trouble, which were characterized by nausea, vomiting, fever and severe headache.

For a week before examination the attack was very severe, high fever, severe abdominal pain; several vomiting spells, evidently projectile in character and not dependent on food intake. He complains of severe occipital headache.

Temperature, 104°. Pulse, 90. Wiry. Marked neck rigidity. Pupils narrow, sluggish reaction to light. Kernig-Brudzinsky, positive. No Babinski; positive bilateral clonus. Knee and abdominal reflexes exaggerated.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, July 11, 1933.

Patient very irritable and hypersensitive, speech incoherent. Heart, lungs, kidneys, showed no pathological changes.

Blood count: Red blood cells, 3,800,000; white blood cells, 20,000; hemoglobin, 70 per cent; polymorphonuclears, 90 per cent; small lymphocytes, 6 per cent; large lymphocytes, 4 per cent.

On examination both ears were discharging very foul smelling thin pus. Destruction of both drum membranes, except for a few small tags. Hearing markedly defective. Hears only when shouted at, and close to his ears.

No nystagmus, no mastoid tenderness or swelling present. No pain produced on percussion of the head. Continuous headache, most marked in occiput and extending into neck.

Spinal puncture resulted in 18 cc., very viscous, milky fluid, containing 4,000 cells per cu.mm., consisting of nearly all polymorphonuclears. Nonne-Appel reaction was strongly positive. Smears show no bacteria. Pressure, 22 mm. mercury. Pressure on right jugular vein increased the pressure to 30 mm. Pressure on left jugular produced 23 mm. Repeated tests gave same results.

There was no doubt that this was a case of spinal meningitis, caused by ear suppuration, but we had to decide which ear was at fault, because the vitality was such that operation on both sides would seriously impair his chances for recovery.

Valuable diagnostic hints were obtained from the X-Rays. Both mastoids showed pathology. The right showed a large area of destruction. The left mastoid was markedly sclerotic. X-Ray diagnosis was bilateral mastoiditis.

The X-Ray findings showing a large cavity on the right side and no cellular structure, only sclerosis on the left side was vital to our decision, which side was most probably responsible for the intracranial complications.

We reasoned that if the large cavity on the right side were filled with pus it would produce edema, or at least tenderness on the right side, because from X-Ray findings it was evident that only a relatively thin layer of bone could separate this big cavity from the skin.

There was no bony destruction of the left mastoid; on the contrary, it was markedly sclerotic. We concluded that the probabilities were that pus under pressure in the left antrum could find an easier

outlet toward the dura from which it was separated only by the thin inner table, as towards the skin from which it was separated by a thick sclerotic mass of bone.

Another important diagnostic aid was the Tobey-Ayres test. A great portion of meningeal complications and one-third of all brain abscesses are caused by sinus thrombosis. Any symptoms which cause us to note sinus complications are valuable in determining the site of the operation.

The Tobey-Ayres test was markedly positive left. This and the X-Ray picture were the only tests pointing to the side which was producing the meningitis, so we decided to operate on the left side.

Semicircular incision behind the left ear was made, to which we added a horizontal incision. The bone was found to be very sclerotic; antrum very small, filled with considerable amounts of granulation and pus. The aditus was very narrow. We opened the bony wall of the sinus and found the sinus wall itself intact. After emptying, the sinus promptly refilled itself.

The tegmen of the antrum was very much necrosed. We removed it and found that a part of the dura behind this necrotic bone was destroyed.

A radical mastoid operation was done and upon enlarging the opening of the tegmen of the antrum we found that the brain behind the necrotic area of the dura was bulging, gray and nonpulsating.

A Cushing trocar was inserted into the brain and at a depth of 2 cm. we found a cavity which contained 2 dr. of thick pus. A rubber drain was placed in the cavity, and we proceeded to enlarge the bony opening, fixing the rubber drain and suturing the horizontal part of our incision.

Postoperative course was very stormy: for one week high temperature prevailed; the meningeal symptoms continued and projectile vomiting persisted several days.

In spite of this, the patient's general condition improved. Temperature dropped slowly, the comatose state resolved itself, and he started to take nourishment. Spinal puncture fourteen days after operation resulted in a slightly turbid fluid containing 800 leukocytes per c.mm. and was free from bacteria.

Twenty-one days after operation, rubber drain fell out, but we had to replace it on account of the return of meningeal symptoms.

Four weeks after operation drain again fell out and was not replaced. The wound healed completely, and after three months the mentality sensorium of the patient returned to normal.

He was able to resume his work and has been working continuously for the past two years.

An interesting finding here was the positive Tobey-Ayres test without any visible sinus thrombosis.

Case 2: On April 1, 1932, a 65-year-old male developed an acute discharging otitis media of right side, which stopped discharging after three weeks of treatment. The drum membrane appeared normal, patient's hearing returned, and nothing pathological could be observed on plane or tip of the right mastoid.

On May 10, 1932, six weeks later, patient developed right-sided headache, accompanied by involuntary spasmodic movements or twitching of his right arm. A competent neurologist was consulted and suspected a brain abscess but did not feel symptoms were important enough to indicate exploration of brain.

For additional consultation patient was sent to the Post-Graduate Hospital in New York City, where he remained from May 20 to June 3, 1932. He underwent a thorough examination by various staff members while there.

Findings: Blood pressure, 180/100. Some albumin and casts in urine. Eye fundi normal.

X-Ray showed some bone destruction as result of suppuration in the ear. No satisfactory findings indicating lesion of brain except some sensory disturbances in path of trigeminus nerve.

Patient was released June 3, 1932, but he did not feel well and had occasional headaches. He had three spells, during which he fell to ground but did not lose consciousness. Once for a short period he lost control of left arm and left leg, and could not close left eyelid. All these symptoms cleared after a relatively short period.

On June 26, 1932, he was brought in to St. Mary's Hospital in Passaic, N. J.

On entrance, his examination revealed unequal dilated pupils and a very sluggish reaction to light and accommodation. The fundi showed marked papillary edema, both sides, more so on the right.

Left side of face, left upper and lower extremity moved very sluggishly.

His reflexes on left side were exaggerated. Hearing fairly good on both sides. There was moderate vascularization of right drum membrane, but no bulging. Landmarks plainly visible.

There was positive neck rigidity. Kernig positive on both sides. Clonus on left side.

Temperature ranged from 99 to 100°. Pulse, 60-64. Blood pressure, 190/120.

Urine showed slight traces of albumin Hyaline casts, urea 40 mgm. Blood count: red blood cells, 3,500,000; leucocytes, 11,000; polymorpho-neutrophyles, 68 per cent s.; small lymphocytes, 24 per cent; large lymphocytes, 6 per cent; monocytes, 2 per cent; hemoglobin, 70 per cent. Blood count on following day showed decrease in leucocytes to 9000.

Spinal puncture recovered 22 cc. turbid fluid under high pressure—40 mm. Globulin markedly increased. Cells per cc., 2500.

Patient was very drowsy and complained of a constant headache in the right temporal region.

There was no doubt in our minds that the patient had a meningitis, probably caused by brain abscess. We decided to do a radical mastoid to determine any possible connection between original lesion and brain abscess.

Thorough examination of mastoid bone, sinus, antrum and tympanic cavity failed to reveal any pathology except slight destruction of bone in tip of mastoid. The dura was found smooth and shiny, but very tense.

To avoid possible contamination, we trephined that part of the temporal area where he complained of severe headache. We made a small puncture opening in the dura. The cerebral liquid was turbid. We inserted a soft catheter for a distance of 1 cc. into the brain and emptied 20 cc. white thick pus out of a large abscess cavity. We left the wound open. Patient lived two days, but never regained consciousness.

This case is especially interesting because of the *long* so-called latent period. As noted above, he had been under observation for two weeks in one of the best hospitals in the country. He did not show any real signs of brain abscess while in the P. G. H.

Case 3: Male, age 27 years. History of discharging right ear for 20 years, never had serious trouble or pain.

Present complaint: For four days had very severe headaches, vomiting, fever, dizziness, and a sensation of falling out of bed.

Physical appearance: Well developed male with normal heart and lungs.

The right ear discharged thin, foul-smelling pus. No drum membrane visible. Tip of mastoid very tender, but not edematous. Heard whispered voice 25 cm. from ear. Bone conduction longer than air conduction. Spontaneous rotatary nystagmus to the left present. Vertigo markedly increased with change of position. Caloric reactions: Cold water, 20° C.; strong vertigo and increased nystagmus after 10 second on right and 45 seconds on left side.

There was no adiakocinesis, no asynergia, no speech disturbance present.

Temperature, 99 to 100°. Pulse, 80. Blood-picture, only 9000 white blood cells. Polymorphonuclears, 84 per cent. Red blood cells, 3,500,000.

Spinal puncture—results 20 cc., very cloudy fluid. Pressure, 20 mm.; globulin strongly positive. Cell count, 3,100. Polymorphonuclears, 75 per cent.

X-ray showed body of right mastoid densely clouded destruction of trabeculae throughout.

Patient suffered quite ostensibly from an acute exaccerbation of a chronic mastoiditis with an acute labyrinthitis just beginning.

As the ear had never been treated before and the hearing was fairly good, I decided to wait for his labyrinthitis to subside. For seven days the patient felt real well, fever subsided, vertigo and nystagmus disappeared, and his appetite returned. He felt very comfortable. On the eighth day temperature suddenly shot up to 104°. Pulse remaining at 80. Patient complaining of a terrific headache, vertigo, and vomiting.

Examination shows classical signs of meningitis, neck rigidity, positive Kernig, hypersensitiveness, irregular, sluggishly reacting pupils. Spinal puncture results—30 cc., very cloudy fluid. Pressure, 30 mm.; globulin strongly positive. Cell count, leukocytes, 3200. Polymorphonuclears, 95 per cent.

We determined to operate on account of meningitis, resulting from an infected labyrinth. Radical mastoidectomy was done. The dura

near the tegmen of the antrum and tympanum did not show any pathologic changes, but the bony capsule of the labyrinth in the area of the lateral semicircular canal was very soft and necrotic. We very cautiously removed the necrotic portion of the labyrinth with gauze and currette, placed a drain, and left the wound open.

Two days later temperature dropped; the neck rigidity disappeared five days later. Immediately after the operation there was a complete peripheral paralysis of the facial nerve, which commenced to clear one week after operation, and returned to normal within twenty-one days.

It is very probable, that in clearing the bony labyrinth capsule, we traumatized the facial canal, without permanent damage to the nerve.

I wish to emphasize that we resorted to conservative methods in treating the meningitis, removing only the infected bony foci.

Case 4: Female child, age 12 years. Had discharging ear for 10 years, post measles. For past two months had severe left sided headaches, recurrent convulsions which usually starts in right arm and leg, spreading to entire body, after which she often sleeps. She was treated for epilepsy because of the convulsions.

Two days before examination the headaches became unbearable, localizing in occiput. Child also had difficulty in moving neck and vomited six or seven times a day.

Examination revealed a well developed child. Left ear discharged a scanty foul smelling pus. Drum membrane almost completely destroyed except for a few tags in upper portion. From behind these tags protruded a gray, white foul-smelling mass with all the characteristics of cholesteatoma. The tip of the mastoid was very tender, as was the mastoid plane.

There was a marked neck rigidity and positive Kernig. Reflexes exaggerated, especially patellar and achilles on the right side. Clonus present. Temperature, 100 to 101°. Pulse, 68. Pupils, normal. Fundi, clear. Urine had a faint trace of albumin. Blood count: leukocytes, 20,000. Polymorphonuclears, 90 per cent. Red blood cells, 3,600,000.

Spinal puncture, 30 cc.; turbid fluid under 31 mm. pressure. Cell count, 2000 leukocytes. Polymorphonuclears, 85 per cent.

X-ray diagnosis was chronic mastoiditis left, with a great amount of bony destruction.

Symptoms suggested a meningitis with involvement of the brain, probable brain abscess on left side. Nearly every classical symptom

of a brain abscess was present: severe headaches, vomiting, Kernig, slight temperature with retarded pulse, spinal fluid under pressure with an increased amount of cells.

The constantly left-sided headaches and convulsions starting on right side seemed to localize abscess to left side of brain, involving the motor area. Exploration of brain seemed almost justified; nevertheless, we decided to follow the old rule, viz., to do a very exhaustive radical mastoidectomy, remove all infected foci, and await developments.

The radical mastoidectomy was easily accomplished because the cholesteatoma had destroyed all the bone. The dura looked healthy except in the area of the tegmen antri, which was a dirty gray and covered with granulations, indicating that the infection started here.

We did not touch the dura. We cleaned out all pathological tissue, did a plastic, drained and dressed the wound.

Course: Recovery without any serious complications. Temperature the first three days was between 101 to 103°. Slight headaches disappeared on fifth day, as also did neck rigidity. No further convulsions. Discharge ceased after five months.

This type of case ordinarily is very tempting for opening of the dura and probing for abscess, but we make it a rule not to explore as long as we find pathology other than brain abscess, which accounts for the symptoms.

In this case the pressure of the cholesteatoma and the subsequent infection of the meninges explained all the general and localized symptoms.

Case 5: Male child, age 12 years. Entered hospital Dec. 22, 1931, with history of a head cold of four weeks duration. Edema of right eye for past two weeks. Copious discharge from right nostril. Occasional severe headaches, which started in the early morning and ceased about 10 A. M. Swelling was intermittent in character.

Child was treated conservatively with adrenalin ephedrine, spray, and ice bag. Temperature, 101 to 104°.

Examination: Continuous discharge of pus from both middle meatuses, more marked on the right side. Marked edematous swelling of right frontal area involving right upper lid.

Skiascopy showed both frontal areas dark.

X-ray diagnosis showed acute frontal sinusitis and ethmoiditis of both sides.

We continued conservative treatment. Temperature varied 99 to 100°. The swelling diminished until Dec. 31, 1931, then suddenly shot to 104°. Frontal regions again started to swell and on Jan. 1, we elicited definite fluctuation. Treatment by medicine was no longer permissible.

On Jan. 2, we incised the abscess along right eyebrow. The medial portion of the anterior wall of right frontal sinus was soft, rough and necrotic, with a fistula leading from sinus to the abscess.

We removed necrotic anterior wall of frontal sinus, enlarging the fistula a little bit, which was filled with foul-smelling pus and granulation tissue. The duct was free and permitted the passage of fair sized cannula from the sinus into the nose. We did not touch the mucosa of the sinus. We placed a drain and dressed the wound. The child felt well for two days after the operation.

On Jan. 4, his condition suddenly became worse. He developed aphasia, distinct neck rigidity, positive Kernig, clonus, and high temperature ranging from 103 to 104°. These symptoms lasted three days, and on Jan. 7, disappeared as quickly as they had come. Temperature was 99°. Pulse regular. Fed himself well, sat up in bed, but had headaches two or three times a day, which were localized to left temporal region.

On Jan. 10, he suddenly became soporous, but could be easily aroused. He lost his ability to talk; he could understand what was said but could not answer. This state lasted for twelve hours.

Jan. 11. He had aphasia which lasted one hour.

Jan. 12. Aphasia lasted two hours. At this time reflexes were exaggerated on right side, with clonus and Babinski, and his right extremity weaker than the left.

Strong neck rigidity and Kernig were present. He vomited several times. Temperature, 102°. Pulse, 90. Blood count, 16,000. Polymorphonuclears, 90 per cent. Spinal puncture, 30 cc., turbid fluid. Pressure, 28 mm. Cells per cm., 800, most polymorphonuclears.

The speech difficulty, the weakness of the right extremity, the clonus, Babinski and the exaggerated patellar reflex on right side all pointed to a meningitis or a left-sided brain lesion.

We decided to check over the operated area. We found that the posterior wall of right frontal sinus was necrotic. We removed the posterior wall and mucous membrane. The dura appeared normal.

We explored the left frontal sinus and found it normal. Then we opened and cleared the ethmoids, which were greatly infected, but we did not open the dura or explore the brain. The wound was partly closed and drains placed.

The healing was uneventful. The aphasia did not recur. The neck rigidity and Kernig lasted about one week. Child left hospital February 6, and has been in good health, free from all symptoms since.

INTERESTING FEATURES.

Case 1: No definite signs of brain abscess preoperatively; nevertheless, we found brain abscess at operation.

Case 2: Was treated at one of our very best institutions and had a very competent examination. In addition, this case was sent to them as a brain abscess, which attracted attention; nevertheless, there were not enough symptoms to require a brain exploration, although later operation proved presence of brain abscess.

In Case 4 all symptoms seemed to indicate presence of brain abscess, both general and localizing signs were apparent; nevertheless, no brain abscess was present.

Meningitis or meningeal irritation were present in all five cases. Four of them recovered. In only one of the cured cases did we have to resort to drainage. In the remaining cases the removal of the pathological bone gave satisfactory results.

SUMMARY.

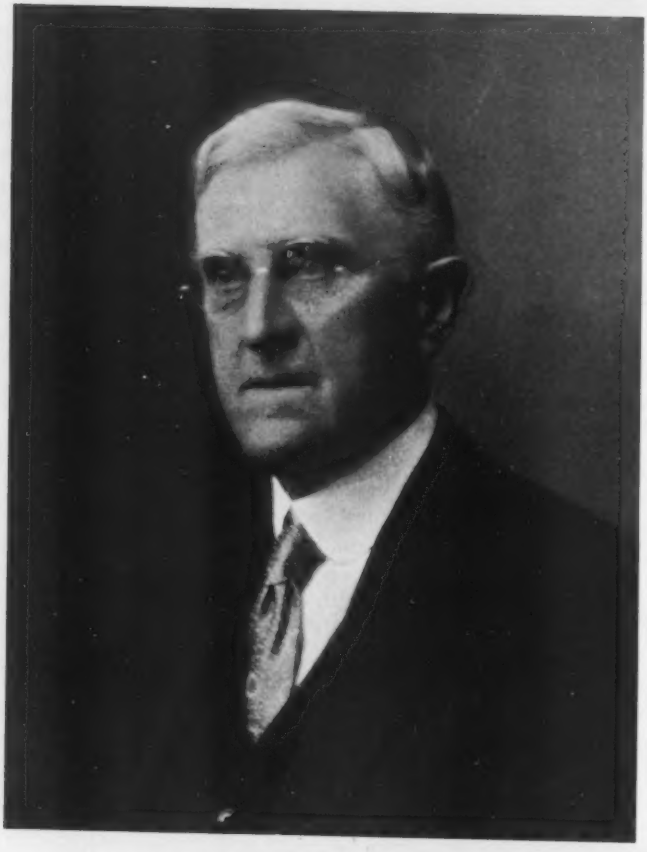
In intracranial complications occurring in the course of ear or nose suppuration, we deem it advisable:

1. To restrict the operation to the side causing meningitis. In bilateral purulent diseases we should use all means to determine the offending ear or sinus.

2. We can use radical procedure in removing all the pathologically changed bone, but we should be conservative in entering the dura. If, after removing pathologic bone the dura looks healthy, it is better to wait. Neck rigidity, Kernig, one-sided clonus, convulsions and paresis are not always absolute signs of brain abscess.

3. If diagnosis of brain abscess is absolute, immediate emptying is advisable.

195 Lexington Avenue.



GEORGE LYMAN RICHARDS.

IN MEMORIAM

GEORGE LYMAN RICHARDS

1863-1923.

The passing of Doctor George Lyman Richards will create a broad gap in the ranks of "The Old Guard" in American Laryngology.

Born in Connecticut, June 1, 1863, he spent his childhood on a farm, receiving his early education in the local grammar and high schools, including some training in a business college, entering Harvard Medical School in the fall of 1883. He received his M.D. degree from Harvard in 1886, served an interneship at the Rhode Island Hospital, in Providence, and began as a country practitioner in the town of East Greenwich, Rhode Island. After practicing medicine until 1893, he went abroad to specialize in otolaryngology; he registered as a student in the University of Holland and, on his return, settled in Fall River in 1896 to engage in the active practice of otolaryngology. He carried out a rather unique professional program in that he maintained offices in Fall River, New Bedford and Taunton.

He was a great traveler, making numerous trips to Europe and on two occasions traveled around the world. In 1919, he went as a member of the Near East Relief Expedition to European and Asiatic Turkey. He was subsequently made medical director of this expedition and in that capacity visited all the relief stations throughout the greater part of Asiatic Turkey. In his world tours he visited many of the important foreign missionary stations in India, China and Japan, where he conducted clinics and engaged in teaching students and giving courses in otolaryngology. During the World War he sat on the Board of Examination of Applicants for Exemption from the Draft.

His religious affiliation was with the Congregational Church. He was President of the New England Otological, Rhinological and Laryngological Society; was President of the American Laryngological, Rhinological and Otological Society; a Fellow of the American Otological Society, and of the American Laryngological Association; a member of the American Medical Association and of his local medical organizations.

To those of us who knew him well his outstanding characteristics were unusual frankness, a fearless presentation of his own convictions; his exceptional ingenuity in instrumentation and technique; a large measure of general information and great familiarity with the literature of his specialties; the gift of a fluent tongue to express his clear and logical thoughts; above all, he was blessed with that most uncommon qualification, "common sense." He was an indefatigable worker and was always reliable in the accomplishment of any task assigned to him in his official positions in the various otolaryngological societies. As Editor of the *Transactions* of the American Laryngological, Rhinological and Otological Society for many years, he performed an important and difficult assignment well.

He was an enthusiastic gardener, a genius with tools and mechanical appliances, and a voracious reader, particularly of books of travel. He was a voluminous contributor to the otolaryngologic literature and was the author of a book entitled, Nose and Throat for the General Practitioner. The large bibliography on otolaryngology which he created by his own contributions is an index of his spirit, his work and his broad-mindedness.

He was popular with his colleagues and many were privileged to call him friend. We last saw him in his home at South Yarmouth, working in his garden, tinkering in his boathouse and reminiscing in his well equipped little library.

He passed away Nov. 9, 1933, at West Dennis on Cape Cod, where he had located his residence during the last three years.

Many close friends and associates will mourn his passing; we will miss his genial personality, his wholesome counsel and his stimulating scientific attitude. One of his greatest joys was the successful development and association in practice with his talented son, Dr. Lyman Richards. To him and to Mrs. Richards we express our heartfelt sympathy. M. A. G.

INDEX NUMBER JAN 9 - 1934

Vol. XLIII DECEMBER, 1933 No. 12

MEDICAL LIBRARY

THE LARYNGOSCOPE

AN INTERNATIONAL MONTHLY JOURNAL
DEVOTED TO DISEASES OF THE

EAR - NOSE - THROAT

FOUNDED IN 1896 BY

DR. MAX A. GOLDSTEIN
Managing Editor and Publisher.

COLLABORATORS:

Dr. ALBERT A. GRAY, Glasgow;
Dr. CHEVALIER JACKSON, Philadelphia;
Dr. F. LABAGNA, Parma;
Dr. M. D. LEDERMAN, New York;
Dr. HARRIS PEYTON MOSHER, Boston;
Dr. FRANCIS R. PACKARD, Philadelphia;
Sir ST. CLAIR THOMSON, London;
Dr. W. A. WELLS, Washington, D. C.

For Contents See Page 1.

Subscription, $6.00 per Annum, in Advance.

Foreign Subscription, 35 Shillings per Annum, Post Free.
Single Copies, 75 cents.

PUBLISHED BY THE LARYNGOSCOPE CO.
912 South Kingshighway St. Louis, Mo., U. S. A.

FOREIGN OFFICE, BAILLIERE, TINDALL & COX
7 & 8 HENRIETTA ST., COVENT GARDEN, LONDON, ENG.

[Entered at the Postoffice at St. Louis, Mo., as Second Class Matter, in July, 1896.]

In Press - - - -

Ready for Delivery

Mail Your Order Now!

PROBLEMS OF THE DEAF

BY

MAX A. GOLDSTEIN, M.D., F.A.C.S.

610 Pages—273 Illustrations—Leather Bound

$6.00 Postage prepaid

An unusual, authoritative and comprehensive volume of XIX chapters, covering every phase of work with the Deaf and Deafened and Speech Defective, by an otologist and teacher of 40 years' experience, including:

Chronological History of Deafness — Anatomy and Physiology — Mechanics of Speech — Speech Defects — Cerebral Localization — Hearing Tests — Methods of Instruction — The Acoustic Method — Training of Teachers — The Deaf Child — Hard-of-Hearing Child — Physician and Deaf Child — Parent of Deaf Child — Deafened Adult — Hearing Aids — Schools for the Deaf — Quacks and Quackeries — Research.

Published by THE LARYNGOSCOPE PRESS

912 South Kingshighway ST. LOUIS, MO.

THE LARYNGOSCOPE PRESS,
912 South Kingshighway,
St. Louis, Mo.

Enclosed please find check for $.....for.....copies of "PROBLEMS OF THE DEAF" by MAX A. GOLDSTEIN, M.D., F.A.C.S.

NAME .....

STREET .....

City ..... State.....

During Pregnancy and the Postpartum Period

supporting treatment is essential. Fellows' Syrup is the most logically prepared tonic at the disposal of the physician.

It contains all the required minerals in correct proportion and in an easily assimilable form. These are Manganese and Iron to renew the blood stream impoverished by continued loss; Calcium to replenish the constant calcium depletion; Potassium, Sodium, and Phosphorus to overcome the neural depression; Strychnine as a tonic to cell metabolism; and Quinine as a gastric stimulant.

There is no better tonic than Fellows' Syrup to the parturient and post-parturient patient. During these trying periods, the suggested dose is one teaspoonful three times daily well mixed with water.

FELLOWS' SYRUP

OF THE HYPOPHOSPHITES

SAMPLES ON REQUEST

FELLOWS MEDICAL MFG. CO., INC.

26 Christopher Street, New York, N.Y.

PILLING TRACHEOTOMY CANNULA SERVICE

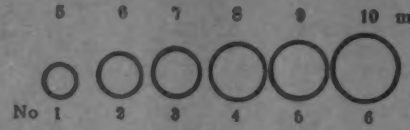
Because of our stock and manufacturing facilities we can promise immediate delivery. You cannot afford to wait.

CHEVALIER JACKSON TRACHEOTOMY CANNULA

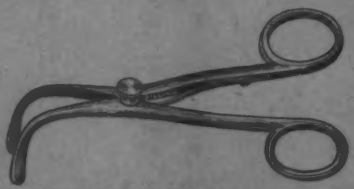
Tracheotomy Cannula, Chevalier Jackson, full curve, regular sizes, 1 to 6, sterling silver, with pilot $7.50
Same, plated $4.50
Tracheotomy Cannula, Chevalier Jackson, cane shape, regular sizes, 1 to 6, sterling silver, with pilot $10.00
Same, plated $6.50

SUGGESTED SIZES FOR TRACHEAL CANNULAE BY DR. CHEVALIER JACKSON

These sizes all leave a by-passage in the trachea for air, so that a cannula (except after total laryngectomy) should never be so large as to fit closely in the trachea.



New-born infant	No. 1
Up to 1 year	No. 1
1 to 3 years	No. 2
3 to 6 years	No. 3
6 to 12 years	No. 4
12 to 20 years	No. 5
Adult (women)	No. 5
Adult (men)	No. 5 or 6



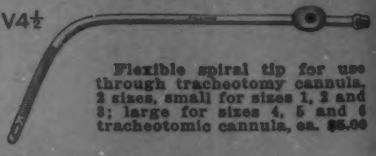
CHEVALIER JACKSON MODIFIED TRACHEAL DILATOR

Correctly shaped, light in weight, not cumbersome. Sizes, regular and infant, each $4.00.

CHEVALIER JACKSON TRACHEOTOMY CANNULA, Larger Sizes

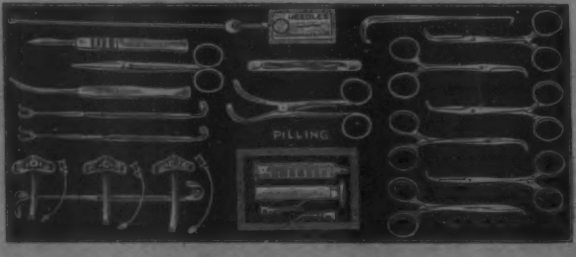
Tracheotomy Cannula, Chevalier Jackson, full curve, sizes 7, 8 or 9, sterling silver, with pilot $9.00

TRACHEOTOMIC SUCTION TUBE



Flexible spiral tip for use through tracheotomy cannula, 2 sizes, small for sizes 1, 2 and 3; large for sizes 4, 5 and 6 tracheotomic cannula, ea. $5.00

EVERY DOCTOR SHOULD HAVE ACCESS TO THIS EMERGENCY TRACHEOTOMY SET OF CHEVALIER JACKSON INSTRUMENTS



Complete set of tracheotomy instruments safely protected in a copper case wrapped in a sterile towel and ready for immediate use; is almost indispensable — it saves time and removes all doubt. Instruments are carefully patterned after the Chevalier Jackson Tracheotomy models, and include aspirating tube, retractors, haemostats, tracheal dilator, scalpel, blunt bistoury, dissecting scissors, thymus retractor, tenaculum, suture needles, catgut and syringe.

With 3 plated tubes $55.00
With 3 silver tubes $62.50

THE GEORGE P. & SON CO. **PILLING** ARCH & 23D STS. PHILA., PA.